FRACTURES
AND JOINT INJURIES

BY

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VOLUME I

Fourth Edition

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HUGH OWEN THOMAS

1834-1891

ROBERT JONES

1857-1933

THEY, WHOSE WORK CANNOT DIE, WHOSE INFLUENCE LIVES AFTER THEM, WHOSE DISCIPLES PERPETUATE AND MULTIPLY THEIR GIFTS TO HUMANITY, ARE TRULY IMMORTAL



HUGH OWEN THOMAS

1834-1891

From the portrait by H Fleury, reproduced by permission of the Curator of the National Portrait Gallery



ROBERT JONES

1857-1933

From the portrait by Frank Copnall, reproduced by the kind permission of Lady Probyn Jones.

PREFACE TO FOURTH EDITION

Once again I must apologise for allowing this book to be out of print. It has happened before, despite unusually large editions running into tens of thousands the demand has always exceeded the supply. This time the delay has been as long as three years and, although there are special reasons, I offer a special apology The old text could, of course, have been reprinted, but to have done so would have denied the advance of surgery—and it would have failed the trust of many whose confidence is a challenge and a disturbance In preparing the fourth edition, every page has been corrected or rewritten, new chapters have been added, and several hundred new illustrations have been prepared There are new chapters on wound-shock and the adaptation syndrome in its relation to shock: on the treatment of open fractures and joint wounds, chemotherapeutic control of infection, early closure of wounds by secondary suture or skin grafting, and plastic surgery in fracture treatment. on transplantation of bone, cancellous bone grafting and intramedullary nailing with a series of case reports of typical bone-grafting operations. and on fatigue fractures and pathological fractures, with a review of bone disease that covers almost the whole of orthopædic surgery

In the second volume there are rewritten chapters on injuries and degenerations of the rotator cuff of the shoulder—as if to recompense for former iniquities in ascribing these lesions to neurosis we are now tending to operate too zealously; on acute and recurrent dislocations of the shoulder—the pathological anatomy and principles of treatment are better understood; on un-united fracture of the shaft of the humerus—it threatens to be the greatest of our failures, on fractures of the upper shaft of the femur—the former problem of prolonged and sometimes permanent disability from outward bowing is being solved by the intramedullary nail; and on fractures of the spine with paraplegia—no longer are the victims of this injury doomed to useless and miserable lives.

A chapter is included on the reactions of bone to metal, based on two Robert Jones Lectures in London and New York, a Hugh Owen Thomas Lecture in Liverpool, and a Fracture Oration to the American College of Surgeons, none of which have been reported before A vigorous attack is made upon the almost universally accepted belief that contact-compression, lag screws, slotted-plates, compression-clamps, and early weight-bearing promote the union of fractures I do not accept a word of it. Forcible compression of bone is pathological rather than physiological, and it avails in the treatment of fractures only in so far as it promotes immobility and protects from shear. In believing this, and denying the view that is held so widely, I reiterate the observations of Hugh Owen Thomas. Moreover, I believe that gaps between the fragments of a fractured bone are always filled if immobility is complete, and that there is never need to make allowance for "inevitable resorption" at the fracture-site Plates and screws do not hold fragments apart, they scarcely hold them at all—certainly not for

more than a few days or weeks. It is indeed unwarranted faith in the capacity of screws to provide sustained fixation of bone that is the cause of non-union of plated fractures, and although stout intramedullary nails have far greater mechanical efficiency than slender screws and plates, they too cause non-union if wholly relied upon to resist angulatory strains, notably, for instance, when nails are driven across the hip joint. Many examples of such failure are quoted in the next text. We should dismiss the phrase "internal fixation of bone"—there is no such thing; there is only internal suture, some sutures being more efficient than others.

I still believe firmly that, apart from interposition of muscle and periosteum, the sole important cause of non-union is inadequate immobilisation; that shearing and rotation stresses are even more harmful than angulatory strains; that compression devices succeed only in so far



It is now possible to break your neck [illegible]

as they protect from such stresses; and that although cancellous surfaces of bone unite soundly and quickly if they are held in close and rigid contact, they still unite soundly, and not much less quickly, if held rigidly with a gap between.

There are two special reasons for unusual delay in preparing this edition. Three years ago it was decided to publish a British volume of the *Journal of Bone and Joint Surgery*, to be shared equally by the [illegible] of the British Commonwealth and the United States of America. With the gracious support of His Majesty the King and the [illegible], the British volume was [illegible] hilarious group of workers, the British volume [illegible] with his vision and wit, Bryan McFarland who [illegible], Philip [illegible] "an illegal venture was threatened with financial ruin", Osmond-Clarke [illegible] miraculously kept us solvent, [illegible] is known throughout the world, Jackson Burrows [illegible] who kept our feet on the ground, Karl Nissen [illegible], Llewellyn Griffiths, Frank Holdsworth, Norman Capener, [illegible]

Roland Barnes, Ernest Nicoll and many others in this country, Bob Harris and Edouard Samson in Canada, Arthur Meehan, Laurie Macdonald and Tom King in Australia, Alex Gillies in New Zealand, and G. T du Toit and Jock Edlestein in South Africa. Together they inspired some of the happiest moments of my life—even including long hours spent working from midnight to dawn Never can I regret this source of delay.

The second cause of delay was my appointment as Arthur Sims Commonwealth Professor of Surgery. In two years I visited nearly every medical school in the British Empire—in Canada, Africa, Australia, and New Zealand. The full inspiration of these visits cannot be measured, the debt owed to surgeons throughout the world cannot be recorded Their influence is reflected in the rewritten pages of this book—but where the reflection may sometimes be dim, the memories of friendship are always vivid.



These men have forgotten their broken backs

There is only one comment I would make on the progress of traumatic and orthopædic surgery in the last decade It does not concern the remarkable advances of technique, medical therapeutics, and laboratory control that have so largely dispelled the fears of virulent infection, the anxieties of surgical shock, and the perils of anæsthesia It concerns quite simply the relationship of the doctor to his patient—the principle of rehabilitation Great progress has been made but still greater progress remains to be made, not perhaps so much in our belief as in our practice, and still more in our teaching "The patient must understand his disability he must regain confidence and be inspired his doubts must not become anxieties, his fears and misgivings must be dispelled, his social problems must be solved, he must be reassured, he must not fear the future" These are words from a former edition, and we all believe them, but within the last ten days I have seen four glaring examples of surgeons destroying confidence, increasing doubt, inspiring anxiety, instilling fear, and promoting misgiving by telling patients that they were neurotic. In not one of them was there

justification for the charge—there was only failure of the surgeon to understand the symptoms and that has been true of scores of similar cases seen in recent years How ready we are when puzzled, or when resentful that a result of treatment is not as good as it might have been, to blame the patient , if the term neurosis is not on the tip of the tongue it is in the forefront of the mind. "Functional disorder" is nearly always the diagnosis of the destitute. It would be better to dismiss the phrase from our vocabulary, and still better to dismiss it from our thought

In other respects there has been important progress in rehabilitation after injury It is now possible to break your neck and enjoy it , treatment is still concentrated not only on the union of fractures but on the function of limbs , there is still emphasis on gymnastics and recreation as well as on surgery and manipulation The miners of Great Britain have superb centres for residential as well as non-residential after-treatment of serious injuries The Royal Air Force has a rehabilitation centre in Surrey just as complete and even more magnificent than any developed during the war In New Zealand, at Dunedin, I saw one of the finest physiotherapy departments in the world, including many gymnasia and treatment-rooms as well as a full-size swimming pool reserved for old ladies and gentlemen recovering from severe fractures and major operations In Great Britain, Canada and the United States there is inspiring success in restoring the victims of spinal paraplegia to useful and happy lives, teaching them to be ambulant with crutches and wheel chairs, to engage in archery and other recreations, and to learn new trades The Disabled Persons Register in Britain totals nearly one million, the vast majority of whom are engaged in normal work, and the rest in sheltered employment Undoubtedly great progress is being made But I still say that in our teaching, and particularly in the teaching of medical students, we should concentrate on the approach of the doctor to his patient Important as recent scientific developments have been it is still true to say that far more patients are cured by the art of medicine than by the science

In writing this book again, as all such books must be rewritten year by year, I owe a debt to many friends throughout the world and particularly to my staff Margaret, Alizon, Blanche, Jacqueline, Marion, Jean, and Walter Robinson my very loyal private assistant who is more than a skilled surgeon —he is a good doctor , to P G Studholme whose knowledge of the English language is superb , to Douglas Kidd who still applies his art to surgery , to W Betts and G Bishop who have checked and corrected all the references ; and to Charles Macmillan and James Parker of E & S Livingstone Ltd , who never fail to understand the eager and sometimes petulant enthusiasm of authors A special debt is owed to Ian Lawson Dick, now of Edinburgh, whose judgment I have learned to respect and whose help has been far greater than I can ever acknowledge in these pages

From Monica and Barrie who grew up with the idea that "Daddy must work" and have now given him up as a bad job, and from my wife whose understanding has been without limit, I ask only for still more forbearance.

R. Watson-Jones

London, 1952.

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PART I

THE PRINCIPLES OF FRACTURE TREATMENT

CHAPTER I

REPAIR OF FRACTURES

A fracture of bone is a rupture of living connective tissue, and its repair is achieved by the cellular growth that characterises repair in all living tissues The training of a surgeon in the treatment of fractures should begin with study of the physiological and pathological reactions of living tissues

HISTOLOGICAL FEATURES

Repair by granulation tissue—During the first few days after injury the histological picture of a healing fracture resembles that of any traumatic exudate undergoing organisation. The exudate is the serum and blood.

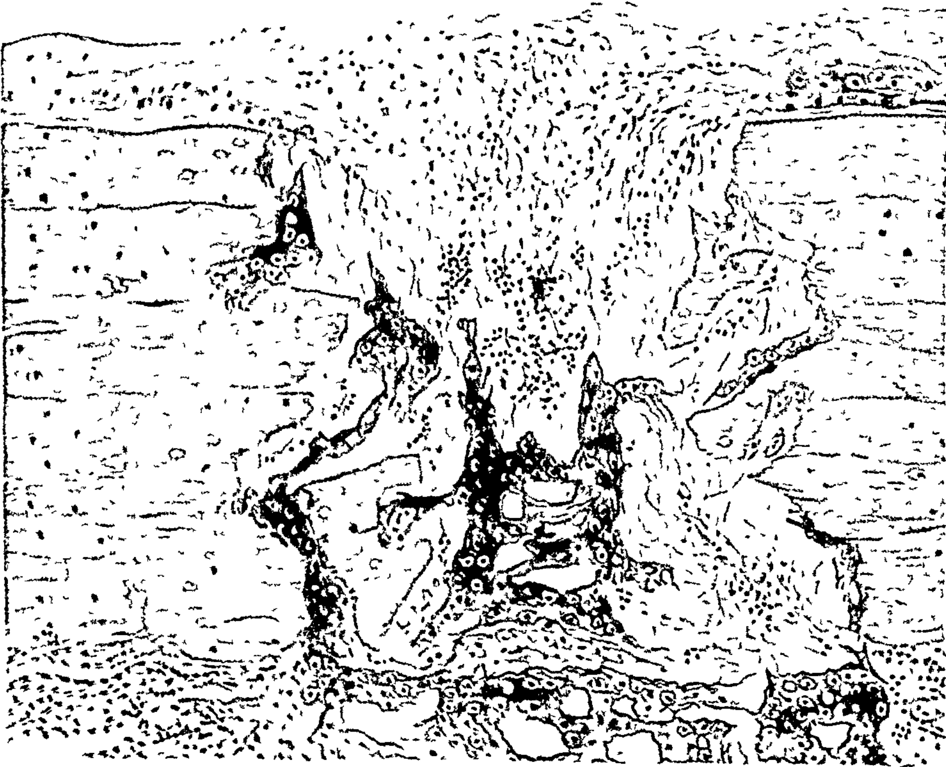


FIG. 1

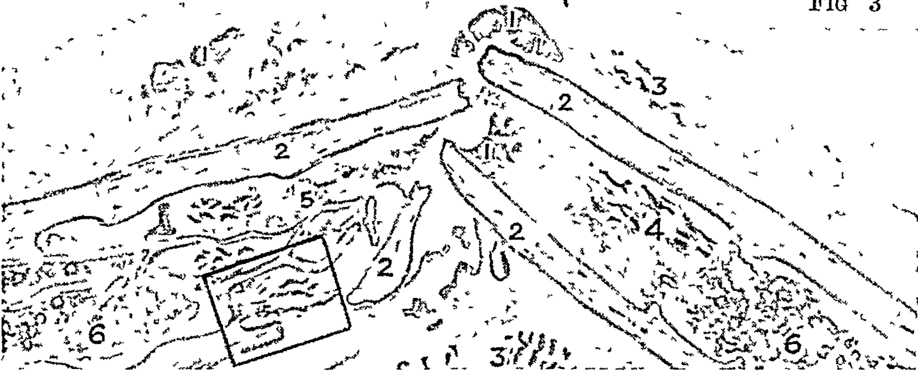
This is no more than a diagrammatic section of a fracture to emphasise that repair takes place by the growth of cells In the treatment of fractures, physiology is more important than carpentry.

partly fluid and partly clotted, between the bone ends, in the marrow, under the raised periosteum and in adjacent tissue spaces Surrounding and invading the hæmatoma is a rapidly growing cellular granulation tissue showing evidence of hyperæmia in the dilated and engorged capillaries.

Fig. 2



Fig 3



Section of fifteen days' old f
in a male aged 40 years (l
A pencil drawing of this
seen in Figure 3, shows—

1, Blood clot.

2, Areas of necrosis of c
with disappearance
bone cells

3, Woven bone formed by p
cells

4, Woven bone formed by r
cells of the marrow

5, Limited area of cartila

6, Marrow cells.

The rectangle indicates th
enlarged × 85 and shown in
4, demonstrating necrosis
corticalis and new forma
woven bone

*(Microphotographs by Mr Kin
Bernhard Baron Laboratories of th
Hospital)*

Fig

Union by primary callus (woven bone)—Within a week, small areas of young bone are laid down around blood vessels, so that the trabeculæ are orientated after the pattern of the vascular tree—an irregular interwoven pattern which gave rise to the name "woven bone." Whereas adult bone is formed slowly in parallel lamellæ corresponding exactly to lines of stress (lamellar bone), this woven bone is formed rapidly around the blood vessels of young connective tissue and thus constitutes the ideal "first-aid" repair of a fracture, to be replaced in due course by mature lamellar bone

The bone is formed equally by parosteal, endosteal and marrow reticulum cells (Figs. 2-4) The conditions under which these various cells usually form bone, but sometimes cartilage and sometimes fibrous tissue, and in varying proportions, is not fully understood But at least it is clear that there is no specific cell, the "osteoblast," which in all circumstances lays down bone. Any cell of the parosteum, endosteum, or marrow reticulum may lay down bone, and on the other hand, if the fracture is not completely immobilised these same cells lay down cartilage Cartilage formation is seen to a limited extent in the repair of every fracture, and as a rule it is transformed ultimately to bone. But when there is mobility of the fragments, as with fractures of the ribs, cartilage formation is increased; and when mobility exceeds a certain limit, as with many fractures in animals, cartilage formation is so vigorous that bone is never laid down—the fracture does not unite and a pseudarthrosis or false joint is established with fibro-cartilage covering the fractured surfaces. Thus on histological grounds alone we see the error of the belief that mobility of the fragments stimulates callus formation and promotes the union of fractures. Repair by granulation tissue, and repair by the woven bone of primary callus, take place with greatest speed and certainty if there is complete immobility.

Consolidation by mature bone (lamellar bone)—The growth of woven bone represents no more than the stage of temporary repair of a fracture, namely, union by primary callus or "clinical union"; final repair and "consolidation" is still to take place Adult lamellar bone can be formed only slowly, and upon a surface of existing primitive bone Every trabeculum of woven bone is removed by osteoclastic resorption and, as it is removed, is replaced by lamellæ in closely parallel arrangement. Excessive subperiosteal new bone is absorbed and the original contour of the bone restored In a variable time, on the average about one year, even the temporarily occluded marrow space is canalised once more, and the fracture may be united so perfectly that its site cannot be determined.

CLINICAL FEATURES

The healing of a fracture is one continuous process, but three histological stages have been defined—repair by granulation tissue, union by primary callus, and consolidation by mature bone—because they correspond to three stages in the clinical history of a fracture. Throughout the first stage the fragments are freely mobile, and the delicate granulation tissue must be protected During the second stage, the growth of cartilage and bone cells leads to increasing stability and the fragments become "sticky" Protection of growing cells from the injury of movement by complete immobilisation of the fracture is still essential The end of this stage may be

judged by clinical tests, there is no longer elasticity or springing, and the fracture is painless when strain is applied. This is the stage of *clinical union* when splints and plaster may often be discarded. Nevertheless reossification is far from complete, and there may be no certain radiographic evidence of union. In the third stage of *consolidation of union* the bone is mature and there is then radiographic as well as clinical evidence that the fracture is united soundly.

BIOCHEMICAL FEATURES

Deposition of calcium in the growing tissue constitutes one of the important distinctions between repair of fractures and repair of soft tissue injuries. In the first few weeks after a fracture there is greatly increased concentration of calcium and phosphorus in the fracture hæmatoma. The general blood calcium level remains unchanged, and the local excess is derived from the bone ends.

1. *Histamine and acetylcholine*—In all injured tissues, liberation of histamine and acetylcholine causes vasodilatation and hyperæmia. Bone reacts to hyperæmia by resorption,[1] and transference of calcium from the fractured ends of the bone to the surrounding fluids continues until the hyperæmia subsides. Only then does reossification begin.

2. *Phosphatase content of the fracture-hæmatoma*—Within a few days the phosphatase content of the fracture-hæmatoma increases to six or eight times the normal level.[2] Phosphatase is a bone enzyme, secreted by proliferating cartilage cells and osteoblasts, and always found where bone formation is most active.[3] It liberates free phosphates by hydrolysis of the organically bound phosphoric acid of the plasma, thus causing supersaturation with calcium phosphate of the fluids bathing the bone.[4] The phosphatase excess persists for about ten weeks, throughout the stage of active growth of cartilage cells and osteoblasts.

3. *Acid tide of the fracture-hæmatoma*—During the first two weeks after fracture, the hæmatoma shows a marked acid tide, the *p*H then swinging back to the alkaline side of neutral.[5] The significance of this tide is not fully understood, but it is clearly associated with local biochemical activity. The repair cannot be accelerated by attempts to raise the blood calcium level by increasing the calcium intake, by alum treatment which controls the phosphorus intake,[6] or by vitamin therapy or endocrine therapy.[7]

PATHOLOGICAL FEATURES

Hyperæmic resorption of bone—Hyperæmia of bone is always associated with porosis, and ischæmia with sclerosis.[8,9] This is seen in osteomyelitis, in neoplasms of bone and after simple contusions or sprains where the bone undergoes temporary resorption. If the fragments of a fractured bone are imperfectly immobilised, shearing and twisting stresses tear the

[1] Watson-Jones, R., & Roberts, R. E. "Calcification, Decalcification & Ossification." *Brit. J. Surg.*, 1943, 21, 461.
[2] Botterell, E. H., and King, E. J. "Phosphatase in Fractures." *Lancet*, 1935, 1, 1267.
[3] Robison, R. "Significance of Phosphoric Esters in Metabolism." New York University Press, 1932.
[4] Tollman, J. P., Drummond, D. H., McIntyre, A. R., and Bisgard, J. D. "Phosphatase Activity in Early Callus." *Arch. Surg.*, 1940, 40, 43.
[5] Stirling, R. I. "Healing of Fractured Bones." *Tr. Med.-Chir. Soc. Edinb.*, 1931, 46, 203.
[6] Helfet, A. J. "Parathyroid Function. Treatment by Aluminium Acetate." *Brit. J. Surg.*, 1940, 27, 651.
[7] Botterell, E. H., and King, E. J. "Phosphatase in Fractures." *Lancet*, 1935, 1, 1267.
[8] Leriche, R., and Policard, A. "Physiology of Bone." London: Henry Kimpton, 1928.
[9] Greig, D. M. "Surgical Pathology of Bone." Edinburgh: Oliver & Boyd Ltd., 1931.

young granulation tissue Repeated traumatisation accounts for recurring hyperæmia, and more and more of the bone-ends undergoes resorption A crack becomes a cavity; a linear fracture becomes a gap fracture. This change is seen most clearly in fractures of the carpal scaphoid bone (Figs. 5-7) Within a few days of injury the fracture may be so fine a crack as



Fig 5



Fig 6



Fig 7

A recent fracture of the scaphoid may be so fine a crack as almost to be overlooked (Fig 5). If it is not immobilised, repeated traumata cause hyperæmic deossification, and the crack widens to a gap (Fig 6) When it is immobilised, reossification takes place and the fracture unites (Fig 7)

to be overlooked in radiographs, it may appear so trivial that there is a temptation to ignore it If the wrist is not immobilised perfectly, the fine crack becomes an obvious fracture within two or three weeks, and a cyst-like cavity within two or three months, the bone-ends showing concave surfaces. If at any stage the fragments are completely immobilised, bone resorption ceases at once, reossification begins, the cavity slowly fills and the fracture unites

If shearing movement continues indefinitely, fibrous tissue is laid down parallel with the fractured surfaces, and there is no continuity between the fragments With the final stage of ischæmic fibrosis, reossification occurs, not in a continuous mass of callus, but in the plaque of bone across the concave ends of the bone fragments When the fractured surfaces become sclerosed, non-union is established.

Infective hyperæmia and the repair of fractures—In a simple fracture which is protected from further injury, traumatic hyperæmia subsides



Fig 8 Fig 9 Fig 10

Infected compound fracture of the tibia, two months, six months and twelve months after injury Provided that complete immobility is maintained, the granulation tissue gradually reossifies and the fracture unites

within about ten days in a compound infected fracture the initial traumatic hyperæmia is followed by even more intense infective hyperæmia which may persist for several months Only when infection is controlled and hyperæmia subsides can reossification begin Treatment of the infection is obviously important Sequestrated fragments of dead bone must be removed as soon as they have separated because, otherwise, persistent low-grade infection delays union of the fracture for months or even years But important as early control of infection may be, continued immobility of the fragments is no less important Destruction of bone by the hyperæmia of repeated movement and strain is no less harmful in infected fractures than in simple fractures Indeed the process of repair is the same, and the

need for complete immobility of the fragments is the same The only difference between the treatment of simple and infected fractures is that protection must continue for many months longer when there is infection. If this is recognised, non-union of infected fractures is no longer a serious problem Sooner or later every infected fracture unites firmly by bone (Figs. 8-10)

RATE OF REPAIR OF FRACTURES

✓ **Influence of age**—In the average uncomplicated fracture there is continuity of granulation tissue in a few weeks, union by primary callus in two or three months, and consolidation of bone in four or five months, but many factors modify the rate of repair. Capacity for the growth of new tissue is greater in the infant than in the adolescent, and greater still than in the adult A fracture of the shaft of the femur in a day-old infant may be united firmly in one month, in a fifteen-year-old youth in two months and in a fifty-year-old man only in three or four months. Malnutrition, cachexia, senile osteoporosis, and deficiency diseases may also delay repair. ✓

Influence of type of fracture—In long oblique and spiral fractures where the marrow cavity is opened widely, there is a large vascular area to promote tissue growth, and union is usually more rapid than in horizontal fractures where medullary callus formation is more limited.

Delayed repair of gap fractures—Union is more rapid if the fragments are impacted into each other than if there is a gap between them. Given time, even considerable gaps may be bridged, but repair is more difficult; there is no natural fixation of the fragments, and complete immobility is more difficult to achieve Moreover, if a gap is the consequence of continued traction, union is still more difficult. Any scar tissue that is subjected to traction becomes weak and attenuated, and, in the case of a fracture, bone formation is discouraged For this reason, the repair of fractures of long bones treated by excessive continuous traction is very slow[12] These factors, however, are responsible only for delay in repair The fracture still unites even despite a gap between the fragments, and despite continued traction, if complete immobility is maintained long enough

Individual variations—Even in fractures of the same type sustained by patients of similar physique and age there may be wide differences in the rate of repair To believe that repair is necessarily abnormal because it is slower than the average is a fundamental mistake. *A fracture is not to be labelled "un-united" simply because union is incomplete within a certain number of weeks or months* It may be following a perfectly normal though slow progress

VASCULARITY AND THE REPAIR OF FRACTURES

One of the most important factors in determining the rate of union is the vitality and vascularity of the fragments If both fragments have a free blood supply, union is rapid, if the blood supply of one fragment is impaired, union is slow if the blood supply of both fragments is impaired, union is very slow, and if one fragment is completely cut off from the circulation, union is very slow indeed.

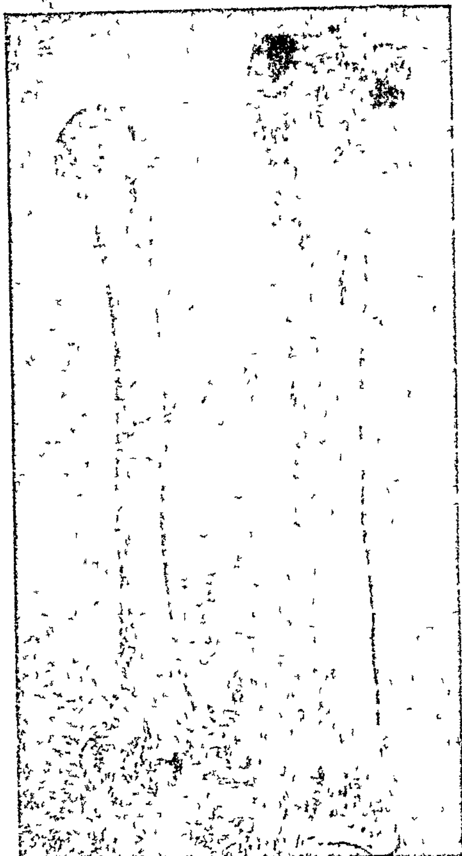


FIG 11

The lower shafts of the humerus and tibia are devoid of vascular foramina The blood supply is entirely from the nutrient artery

Importance of free blood supply—Rapid growth of the granulation tissue that initiates healing of a fracture demands a free blood supply and even, for the first few days, an actual hyperæmia. The blood supply of normal bones is derived not only from the main nutrient vessels but from many other vessels entering the cortex through capsular, ligamentous and tendinous attachments.[1] At the ends of long bones in the region of synovial reflections and joint capsules there are scores of vascular foramina Through these foramina there pass not only veins from the bone but also arteries to the bone. Indeed in growing children this metaphyseal area is more richly supplied with blood than any other region. Even in the adult, the ends of long bones have a very free blood supply and fractures unite rapidly. Fractures of the neck of the humerus, supracondylar fractures of the humerus, fractures of the neck of the radius. Colles' fracture of the radius, supracondylar fractures of the femur. fractures of the tibial tuberosities and fractures just above the ankle joint are all characterised by rapid union Similarly, the innominate bone which has many ligamentous attachments and shows vascular foramina on all its surfaces is freely supplied with blood and the repair of fractures is again rapid

Impaired blood supply of one fragment—On the other hand, the lower third of the shafts of the tibia, the humerus and the ulna may be entirely devoid of vascular foramina The bone in these regions depends mainly for its blood on the nutrient artery (Fig 11) A fracture of the shaft may cut off this source of supply from the distal fragment, so that the vitality of this fragment is impaired, its contribution to the growth of granulation tissue and callus is feeble, and union is slow (Figs 12-13) This difference in the vascularity of the two fragments may be a striking feature of operations performed for non-union of fractures of the lower

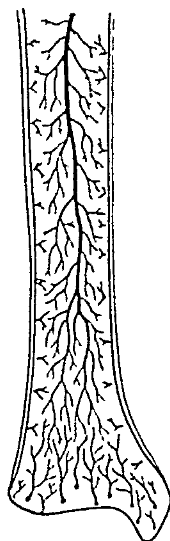


FIG 12

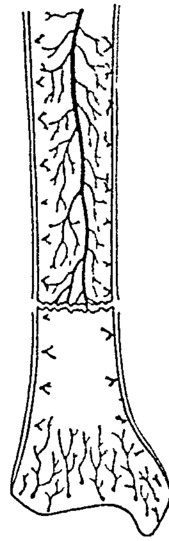


FIG 13

A fracture of the lower shaft of the tibia cuts off the blood supply of the nutrient artery from the lower fragment Union is therefore slow

[1] Johnson, R W "Physiological Study of the Blood Supply of the Diaphysis" *J Bone Joint Surg*, 1927, 9, 153

shafts of the humerus, ulna and tibia Whereas the bone of the proximal fragment bleeds freely, the distal fragment may appear to be almost exsanguinated In these cases the poor blood supply has been responsible for slow union, and failure to prolong the period of immobilisation accordingly has been responsible for non-union

Impaired blood supply of both fragments—Figure 14 is the radiograph of a double fracture of the shaft of the tibia. Both fractures were sustained simultaneously The treatment of the two fractures by manipulative reduction and immobilisation in an unpadded plaster cast was identical. Nevertheless the rate of repair was very different. The upper fracture was



FIG. 14

Double fracture of the lower shaft of the tibia At the upper fracture the blood supply of one fragment is impaired At the lower fracture the blood supply of both fragments is impaired.



FIG. 15

Union at the upper fracture was somewhat slow and was complete only after five months Union of the lower fracture is very slow indeed, and it is still incomplete even after eleven months.

consolidated firmly in five months Figure 15 shows the radiograph after eleven months of continuous immobilisation Repair of the upper fracture is so complete that practically all trace of the injury has disappeared, and yet the lower fracture is still far from firmly united. What is the difference in the blood supply of the two fractures? The upper fracture has a normal blood supply on the proximal side, and an impaired supply on the distal side which has been cut off from the nutrient artery. Union, therefore, was rather slower than normal The lower fracture has a poor blood supply on both sides; even the proximal fragment has in this case been deprived of its blood by a second fracture higher in the shaft Since neither fragment has a sufficient blood supply, neither is capable of promoting vigorous granulation tissue growth and a bone-grafting operation was necessary before firm union was secured

Complete loss of blood supply to one fragment—If the blood supply is entirely cut off from one fragment so that it is completely avascular, the

fragment remains inert and can take no part in the process of repair This occurs in certain fractures of the neck of the femur, the carpal scaphoid and other bones. There is radiographic evidence of this avascularity, for the dead bone cannot be resorbed or lose its calcium content so that it appears more dense than the adjacent living bones which undergo the progressive porosis of disuse and hyperæmia. If one fragment takes no part



Fig. 16



Fig 17



Fig 18

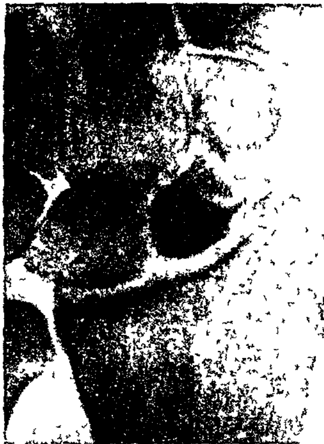


Fig. 19



Fig. 20



Fig 21

Dislocation of lunate and half scaphoid, with complete loss of blood supply to the proximal scaphoid fragment (proved by its apparent density—see Chapter V) Despite this, radiographs two months, six months, ten months, fourteen months and eighteen months after injury show steady invasion of the avascular fragment and ultimately union of the fracture

at all, it is obvious that repair must be very slow indeed The relative indolence of cell proliferation is shown by lack of the excess local phosphatase[1] which is characteristic of all other types of fracture (p 6). But although repair is slow, it can still be accomplished if immobilisation is suitably prolonged The living fragment promotes tissue growth which slowly invades and ultimately replaces the dead bone The process may occupy eighteen months or even two years, and immobility of the fragments must be maintained throughout this time (Figs 16-21)

[1] Botterell, E H , and King, E J *Lancet*, 1935, 1, 1267.

CHAPTER II

DELAYED UNION AND NON-UNION

"*We do not expect union of bones if motion of the fragments is permitted.*"
—HUGH OWEN THOMAS, 1834-91

"*The speed of reunion of bone is in direct ratio to the rigidity with which the two pieces are placed together*"—RICHARD VON VOLKMANN, 1830-89

Non-union of fractures is due to the failure of surgeons much more than to the failure of osteoblasts. With few exceptions it is an avoidable complication It has been customary to enumerate many etiological factors such as imperfect apposition of fragments, interposition of soft tissues, distraction of the fragments, impairment of blood supply, functional disuse infection of bone, osteoporosis, senile change, operative interference, stripping the periosteum, plugging the medulla reaction to plates and screws, inhibitory effect of synovial fluid, lack of blood clot between the fragments and compression of the fracture-hæmatoma by an unpadded plaster cast But these conditions, although some of them may perhaps influence the *rate* of union are not the cause of *non*-union There is only one cause of non-union of fractures with a continuous hæmatoma between the fragments—

the cause of non-union is inadequate immobilisation.

The rate of repair has already been discussed Age makes the difference between union of a fractured femur in three or four weeks in the infant and three or four months in the adult, but at all ages, even over the age of ninety, fractures of the femur unite. Impairment of blood supply may delay repair, but if fractures showing this delay are protected long enough they still unite Operative exposure of a fracture may reduce the blood supply by stripping soft tissue attachments from the bone, but if post-operative immobilisation is adequate, plating and other operations do not cause non-union Excessive traction causes striking delay in the healing of fractures, but union still takes place if immobilisation is prolonged Even severe infection of a fracture is not a cause of non-union if immobilisation is maintained long enough

Age, constitution, blood supply, infection, type of fracture and method of treatment make union difficult or easy, slow or rapid If these conditions are favourable, as, for example, in fractures near joints where bone is vascular and cell growth vigorous, a fracture may unite despite imperfect immobility But if conditions are unfavourable and cell growth is slow and difficult, the fracture unites only when immobilisation is adequate and prolonged. The cause of non-union in gap fractures, infected fractures, plated fractures and fractures with poor blood supply is not the gap, the infection, the plating or the blood supply, but the failure to recognise that these factors cause slow union, and the failure to prolong immobilisation accordingly.

In a series of 800 consecutive fractures of the shafts of the femur and tibia which were investigated,[1] there was a high proportion of comminuted,

[1] These cases are analysed in detail in Volume II

contaminated and severely infected fractures; many different methods of treatment had been used including manipulation and plaster, skeletal traction, open reduction and internal fixation. Whatever technique had been employed, there was always insistence on continued and uninterrupted immobilisation until repair was complete. In the whole series *there was not a single case of non-union*. Many fractures were slow in uniting, but not one fracture failed to unite.

DIFFERENTIATION OF SLOW UNION, DELAYED UNION AND NON-UNION

Formerly, a certain number of weeks was fixed as the period required for the union of a fracture in each region—clavicle, three weeks; scaphoid four weeks; tibia, eight weeks; femur, ten weeks; and so on. More recently the periods have been extended. Three months is now granted, or

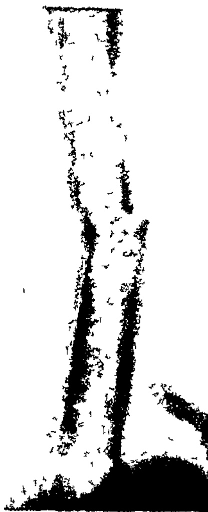


FIG. 22



FIG. 23

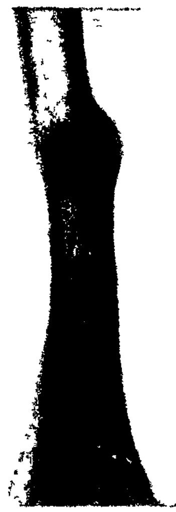


FIG. 24

Slow union

Fracture of the humerus—three, six and twelve months after injury. There is indolence and slow union, but no cavitation or non-union.

even four months; but the fundamental error is still made of fixing a period at all. The dictates of a calendar are accepted, and every fracture not united within the specified time is labelled "un-united." A new regime of treatment is then instituted: the plaster is removed; heat and massage are employed; hammering and damming[1] are prescribed; a walking calliper splint is fitted; and immobilisation is necessarily suspended. It would be as reasonable for a gardener to uproot every plant not in flower by a specific

[1] **Hugh Owen Thomas' Hammering and Damming.**—The "hammering and damming" of Hugh Owen Thomas consisted in percussion with a rubber hammer, and passive venous congestion by means of a tourniquet, repeated two or three times a day. But a study of "Contributions to Surgery and Medicine," vol. 6, June 1886, [illegible] In reporting one such case he wrote:

"During the treatment of this case I made one very important omission, the use of which would have shortened the time of repair. ... No fixation of the fractured bone was attempted. The fact that repair could best be conducted in combination with efficient fixation I had not divined ... nay, I rather inclined to the belief that fixation was always a means of restoring symmetry at the cost of delaying repair, an error of theory thus leading me to defective treatment."

Not only does this observation show that whatever merit there may or may *not be in passive congestion*, Thomas recognised that continued immobilisation was fundamentally more important, but also it is the answer to those [illegible] who have thought that fixation is "*a means of restoring symmetry at the cost of delaying repair*." The fallacy of this view, and the defective treatment to which it leads, was declared sixty years ago.



Fig. 31
One day



Fig. 32
Three months



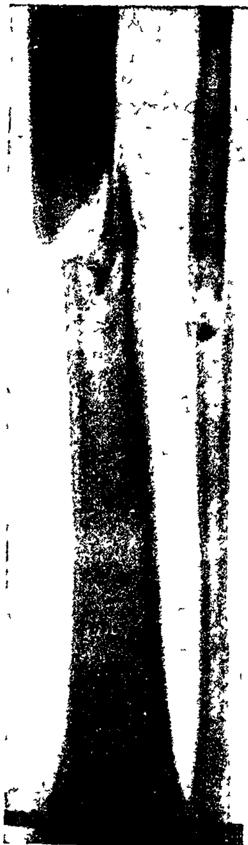
Fig 33
Nine months.



Figs 34-35
Twelve months.

Slow union due to poor blood supply

Fracture shaft of tibia with slow union from poor blood supply of the distal fragment The distal fragment showed relative density (Figs. 32 and 33) which disappeared gradually as the bone was revascularised from below. (See also Figs 533-534, p 323)



Ten days

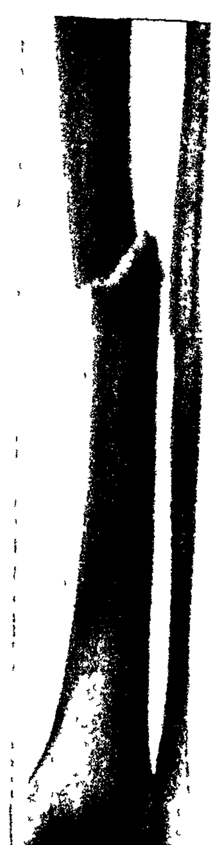


Twenty days



Three months





Six months

Slow union due to distraction

Figs 36-40—Distraction due to excessive pull was corrected on the twentieth day, but the harm had been done There is typical indolence and slow union



One day.



One month



Eight months



Thirteen months

Slow union due to distraction

Figs. 41-44—Distraction continuing for many weeks is still more serious In this case there has also been inadequate immobilisation as shown by heaped up bone round the fracture

sound If immobilisation had ceased at an earlier date, the fracture would have failed to unite. Slow union would have become non-union. The non-union would not have been inevitable as so often is assumed, it would have been avoidable.

Slow union of this degree is seldom seen in fractures of the shafts of long bones not complicated by infection, excessive traction or open operation, but it is significant that the normal process of repair can sometimes last as long as twelve months In fractures of the carpal scaphoid bone and fractures of the neck of the femur, impairment of blood supply occurs more often (Figs 28-30). An accurate forecast of the necessary period of immobilisation cannot be made The fracture of the scaphoid with complete interruption of blood supply, illustrated in Figures 16-21, united only after eighteen months Similarly, in fractures of the neck of the femur, experience gained by the nailing operation shows that complete repair may take twelve months or even longer.

SLOW UNION FROM EXCESSIVE TRACTION

Slow union of fractures has occurred with increasing frequency in recent years. There can be little doubt that this is associated with the higher standard of accurate reduction and end-to-end apposition now demanded, and particularly with the methods of skeletal traction employed in achieving this reduction The danger does not lie in the correction of over-riding bone fragments by neutralising the elastic recoil and spasm of muscles, but in over-correction, in excessive pull which separates the fractured surfaces and distracts the fragments, and especially in over-correction continued for several days or weeks by the suspension of heavy weights from a skeletal traction pin.



FIG. 45
Ten weeks



FIG. 46
Eight weeks.

Rapid union when traction is avoided

Compare the rapid union of spiral and oblique fractures (similar to those in Figs 36-44) when no traction is employed, and redisplacement is prevented by the internal fixation of a transfixion screw.

The danger of skeletal traction—Powerful traction can be maintained from a pin driven through the bone and attached to a weight suspended over a pulley at the foot of the bed By using weights of 25 to 30 pounds it is easy to cause overlengthening or distraction. In compound fractures, especially in war injuries where the muscles surrounding the bone are severely damaged, muscle weakness may be so profound that distraction is produced by as little as a weight of 10 pounds Moreover, there is often such loss of tone that when the fragments are once distracted they

are not pulled together again even when the weight is reduced, and the gap persists The bridging of such a gap offers unnecessary difficulty in the repair of a fracture But the delay is much greater than the time taken in merely bridging a gap Many months are added to the necessary period of immobilisation by only slight separation even when rapidly corrected (Figs 36-40). If the fragments are distracted at the most dangerous period not within a few days of injury but after several weeks when the fracture hæmatoma is organising and being replaced by cellular tissue, a fracture that would otherwise unite in two or three months unites only after ten or twelve months. It behaves exactly as if the blood supply had been seriously reduced. It is probable that this is what in fact occurs, and that slow union after excessive traction is due not only to the obvious tearing apart of young cellular layers but also to the strangling by tension of capillaries in the organising hæmatoma, so that indolence continues even after distraction is corrected. It is noteworthy that the effect is most marked in the middle third of the tibia where the blood supply is already poor, and less evident in a vascular bone like the femur. The delaying influence is least obvious in the shaft of the fibula



FIG 47

Example of grossly excessive tibial traction Not only was the limb over-lengthened, and union seriously delayed, but the knee joint ligaments were severely stretched and the cruciate ligament was actually avulsed with a fragment of bone

The greater danger of traction without immobilisation—There is even greater danger when traction is used to control alignment as well as to prevent over-riding, the method adopted by Bohler in treating fractures of the shaft of the femur, and by some surgeons in treating fractures of the tibia No local splint or plaster is used, the limb rests on a simple cradle and the weight alone maintains length, alignment and apposition. It is often found that weight sufficient to prevent angulation causes distraction of the fragments, and if the weight is reduced to prevent distraction, angulation recurs (Figs 48-52). The temptation to employ excessive traction is almost irresistible Moreover, to the delay of excessive traction is added the delay of inadequate immobilisation This technique has been responsible for many cases of non-union, particularly in fractures of the shaft of the femur. Skeletal traction must be used with caution The practice of attempting gradual reduction of displacement over a period of many weeks by continued heavy traction on the limb should be abandoned. The object of treatment should be to correct displacement at once, within a day or two of injury, and then

FIG. 48
Six weeks

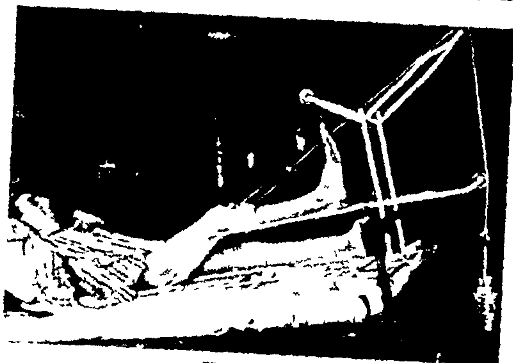

FIG 52

FIG. 49
Twelve weeks.

FIG 50—Fourteen weeks.

FIG. 51—Fourteen months.

Slow union due to distraction

Excessive pull continuing for six weeks caused distraction of the fragments (Fig 48) Even when the weights were reduced this was not corrected (Fig 49) Still further reduction of the weight allowed angulation to recur (Fig 50) Finally a bone-grafting operation was necessary (Fig 51) Excessive traction without immobilisation causes slow union and deformity.

2 A

to immobilise in the corrected position with the least possible traction and certainly no more than is strictly necessary to prevent redisplacement. In the tibia it is better to prevent redisplacement of an unstable fracture by a transfixion screw buried in the bone than by a traction pin and suspended weight

The danger of skeletal transfixion—Another technique, developed in the United States, is to drive one or more pins through each fragment, reduce the displacement by mechanical means, and incorporate the pins in a plaster cast or in a special apparatus that locks them together and prevents approximation If the fragments are fixed in distraction, with a slight gap between the fractured surfaces, union is very slow The sequel is exactly similar to the slow union of excessive skeletal traction.

DELAYED UNION FROM INADEQUATE IMMOBILISATION

Two purposes are served by the immobilisation of a fractured bone (i) control of position, which prevents displacement of fragments and mal-union, (ii) protection of growing cells, which prevents delayed union and non-union Splints or plaster adequate for the first purpose may yet be inadequate for the second All the injuries illustrated in Figures 53-55 may appear to be immobilised adequately The fracture of both bones of the leg is protected from angulation and deformity by a plaster cast to the knee, and still more certainly by a plaster to the mid-thigh, but neither of these plasters controls rotation movement of the upper fragment of the tibia Even the mid-thigh cast allows rotation of the femur to be transmitted to the upper fragment of the tibia, because the knee joint is fully extended Unless this rotation movement is prevented, there will be delayed union or non-union The short forearm plaster may prevent gross displacement of the fracture of both forearm bones (Fig 55), but the cast is not controlling supination and pronation movement and this movement is accompanied by rotational strain on the fracture of the ulna which causes non-union Neither the cock-up splint nor the plaster cast used for a fracture of the scaphoid is controlling wrist movement fully (Fig 53), and although the fragments of the scaphoid will not displace, the fracture will certainly fail to unite. At one time the degree of immobilisation illustrated in these figures was accepted as classical treatment At that time non-union was so common that lists of causes were enumerated to explain it Now that the need for protection of growing cells by more complete immobilisation is recognised, non-union of these fractures does not occur. Protection of the growing cells demands (i) adequate immobilisation, (ii) uninterrupted immobilisation, (iii) immobilisation for an adequate period, (iv) complete immobilisation until union is sound.

Adequate immobilisation—As a rule the joints above and below the fracture must be immobilised even although less complete fixation would suffice to prevent displacement The special precautions needed to protect fractures from rotational and shearing strains are discussed in later paragraphs

Uninterrupted immobilisation—The immobilisation must be continued without interruption If the only object was to prevent deformity, movement of the fragments at the time that a plaster was being changed would be

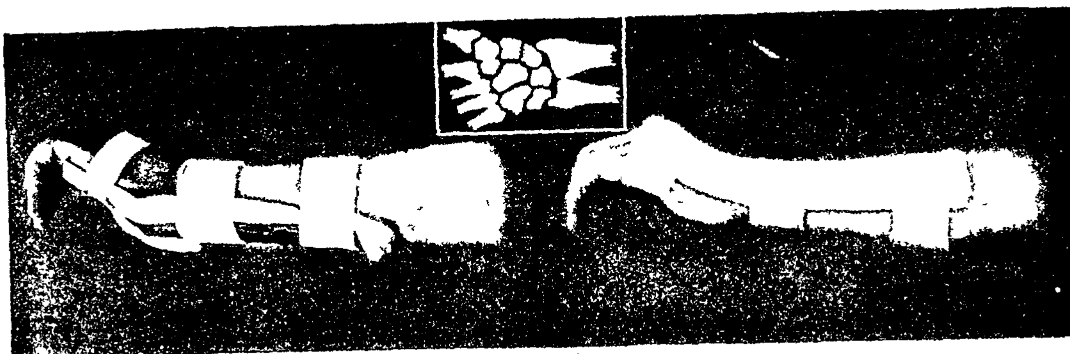


FIG. 53

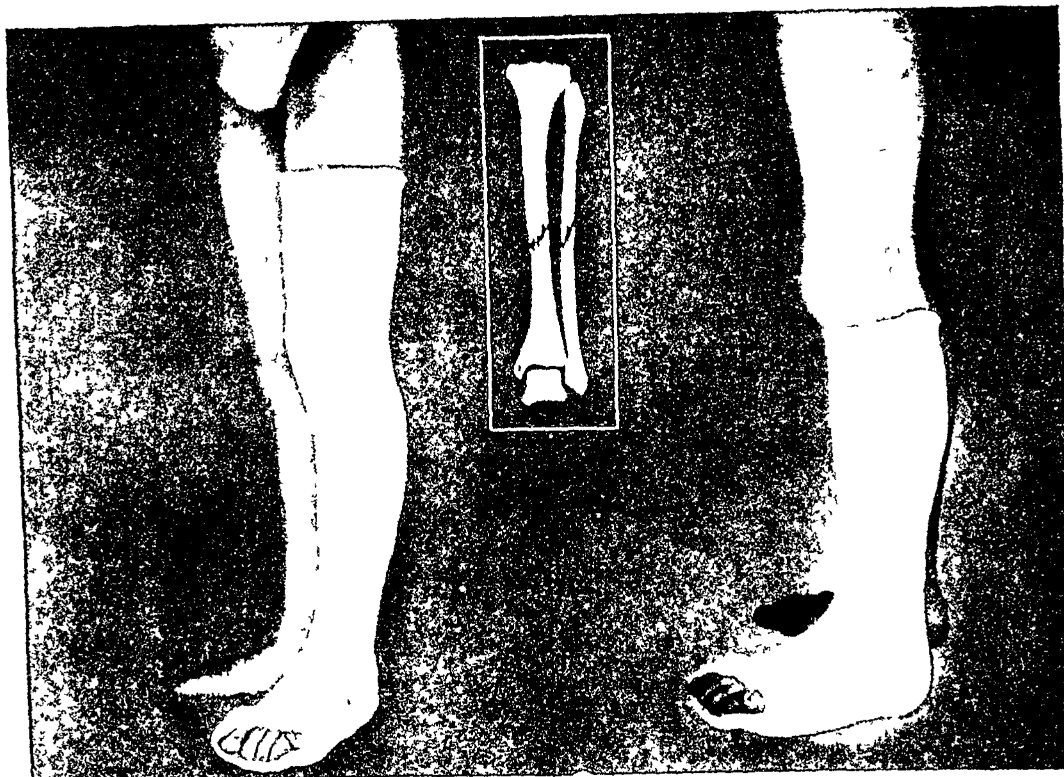


FIG 54

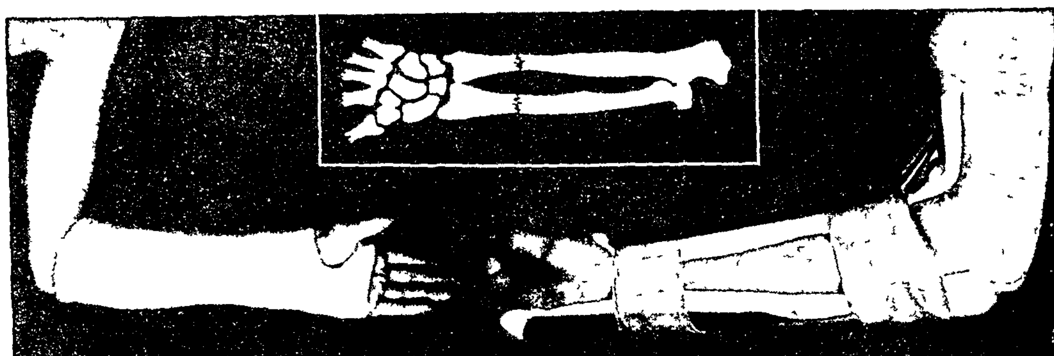


FIG 55

The cause of non-union of fractures

Fig. 53—Fracture of carpal scaphoid bone Fig 54—Fracture of shafts of tibia and fibula.
Fig. 55—Fractures of shafts of radius and ulna

In every case the immobilisation is inadequate Although the splints and plaster that have been used may suffice to prevent deformity, they do not protect the growing cells completely. There will be delayed union or even non-union.

unimportant so long as the position was finally adjusted. In fact, however, every single movement sets back the process of repair. If a new plaster is to be applied there must be no interval when unguarded movements are permitted The discarded plaster must be removed by the surgeon himself in the theatre, not by a nurse in the ward or by an assistant in the anteroom The surgeon must support the limb, protect the fragile tissues, and never permit careless handling and strain which may even amount to refracture. It is obvious that union of a fracture can never take place if refractures are sustained at regular monthly intervals, every time the plaster is changed.

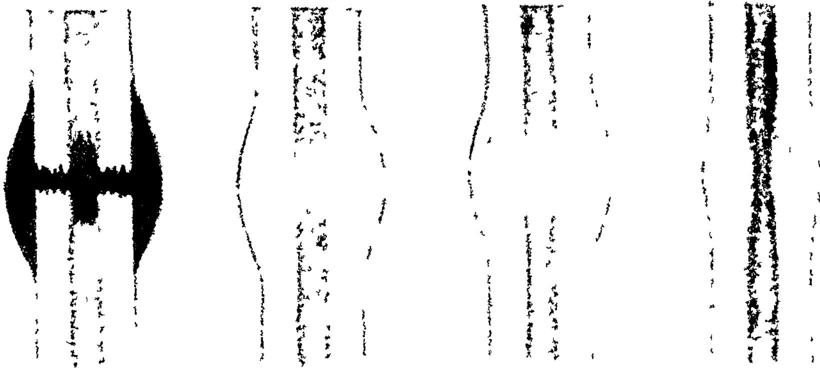


FIG 56

Healing of a perfectly immobilised fracture The fragments are joined by an uninterrupted growth, in the early stages of granulation tissue, and in the later stages of callus Continuity is established.

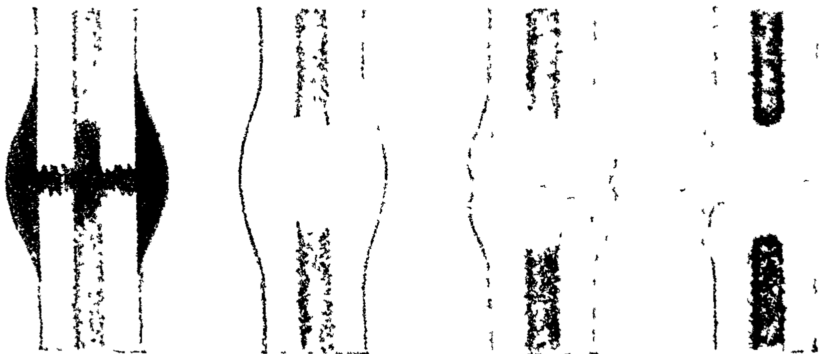


FIG 57

Healing of an imperfectly immobilised fracture Shearing and rotation strains create a plane of cleavage Continuity is interrupted and fibrous tissue is laid down parallel with the fractured surfaces

Immobilisation for an adequate period—Immobilisation must be continued until the fracture is united, however long that may be—twelve weeks in the average case, four months in some cases, and six to twelve months or even longer in exceptional cases There is no fixed number of weeks for the repair of any fracture If every fractured tibia is taken out of plaster at the end of eight weeks, all slowly repairing fractures fail to unite If the arbitrary period is fixed at ten weeks, or at twelve weeks, the incidence of non-union is lower, but there will still be failures Immobilisation must be continued, regardless of time until there is clinical evidence of sound union.

Complete immobilisation until union is sound—It is not enough to immobilise a fracture completely for eight weeks, or ten weeks, or

twelve weeks, and then to "protect" it by some less adequate form of immobilisation—for example, in a fracture of the tibia by changing from a full-length plaster to a below-knee plaster. If union is already sound, such protection is unnecessary; whereas if union is not already sound, inadequate immobilisation induces traumatic hyperæmia once more and the callus, which has begun to calcify, undergoes resorption (Figs 63-66)

The dangers of rotation and shearing strains—The type of movement most harmful to the repairing fracture is that caused by a rotational or shearing stress which creates a plane of cleavage between the fragments (Figs 56-57). Such movement encourages the formation of cartilage and fibrous tissue in the space between the bone-ends, and the fibro-cartilaginous tissue is laid down in the plane of movement, parallel with the fractured surfaces This type of rotational strain is clearly evident in all the regions where non-union was so common in former days—the lower shaft of the ulna, the lower shaft of the humerus, the lower shaft of the tibia, the waist of the scaphoid and the neck of the femur



FIG 58

Un-united fracture of the ulna

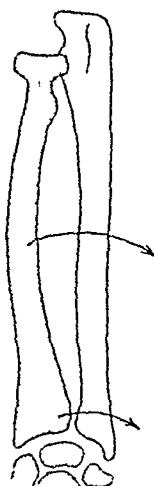


FIG. 59

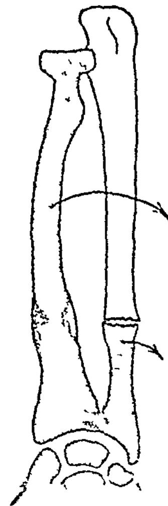


FIG. 60

A forearm plaster does not prevent supination-pronation movement, and the resulting torsion of the fragments of the ulna causes non-union.

Rotation strain in fractures of the shaft of the ulna—When fractures of the shafts of both forearm bones were treated by means of a pair of gutter splints from the level of the hand to just below the elbow, the fracture of the radius usually united without difficulty but the fracture of the ulna remained un-united (Fig 58). The splints prevented angulatory movement of the fragments, but they did not prevent pronation and supination of the forearm. This movement is a bucket-handle swing of the radius round the fixed axis of the ulna. The two fragments of the radius swing together and there is no torsional strain; the fragments of the ulna rotate on each other and the torsion prevents union (Figs. 59-60) So common was this error of treatment that fractures at this level were regarded as particularly susceptible to non-union Indeed this was chosen as the site of election for arthroplasty of the forearm in cases of radio-ulnar ankylosis. It was believed that if the lower shaft of the ulna was divided by osteotomy the formation of a false joint was almost inevitable The fact is, however, that if the forearm is properly immobilised by means of a plaster cast from the hand to the upper arm, so that radio-ulnar movement is arrested completely, fractures of the lower shaft of the ulna always unite firmly.

Union is often slow It may be necessary to continue immobilisation for four months or even longer, and a complete plaster to the upper arm must be retained throughout this time Surgeons must not be tempted to replace the complete plaster by a short forearm cast at the eighth or tenth week from fear of elbow stiffness *A fracture of the ulna is subjected to greater*



FIG 61

Most fractures of the shaft of the humerus show vigorous callus formation and rapid union despite the obviously incomplete immobilisation of simple gutter splints

rotational strain in a short forearm plaster than in no plaster at all Movement at the superior radio-ulnar joint is not limited by such a plaster and pronation and supination are still possible, but close moulding of the plaster round the wrist obstructs movement at the inferior radio-ulnar joint Rotation occurs more easily at the unsoundly united fracture of the ulna than at the stiffened inferior radio-ulnar joint. The fracture of the radius may go on uniting because there is no torsional strain, but bone apposition at once ceases at the fracture of the ulna. Resorption takes its place, callus already developed is absorbed and non-union results

Freedom from shearing and rotation strain in fractures of the ribs—The ease with which fractures of the ribs unite despite constant respiratory movement was claimed to support the old fallacious view that slight movement encouraged union. But respiratory movement of the ribs is exactly analogous to the bucket-handle swinging of the radius Both rib fragments move together, and the intercostal fibrous and muscular tissues provide a perfect natural protection from shearing and rotation strain

Rotation strain in fracture of the shaft of the humerus—A fracture of the shaft of the humerus is immobilised completely only if both elbow and shoulder joints are fixed by means of a plaster spica Nevertheless most fractures of the humerus are characterised by such vigorous callus formation that union takes place within five or six weeks even despite incomplete immobility. Short gutter splints, or a plaster slab, are sufficient , and if the wrist is suspended from the neck by a collar-and-cuff sling, mild traction is maintained by the weight of the limb (Fig 61) Occasionally, however, fractures in the lower shaft are of exactly the opposite type Clinical tests at the sixth week show that the fragments are still freely mobile and union has scarcely begun. These are examples of slow union due to injury of the nutrient artery and impairment of the blood supply The same slow union may result from excessive traction



FIG. 62

Indolent fractures of the shaft of the humerus with slow union will not unite at all immobilisation is incomplete The shoulder and the elbow joints must be completely immobilised by means of a plaster spica.

The surgeon must be on guard. The short splints and sling which suffice for other fractures of the humerus are entirely inadequate for the indolent fracture If they are relied upon the fracture will fail to unite (Fig 62) Every movement of the forearm and hand from the chest wall rotates the lower fragment of the humerus and strains the cellular growth which is already so feeble. Firm union is secured only if the fracture is protected from every rotation strain by the complete immobilisation of a plaster spica.

This complete immobility must sometimes be continued for many months. The harmful effect of even delayed rotatory movement is proved by the case shown in Figures 63-66 Union was characteristically slow Despite



Fig. 63 Fig. 64 Fig 65 Fig 66

Fracture shaft of humerus, three, five, seven and twelve months after injury. After five months' immobilisation, union was almost complete (Fig 64). An unpadded plaster to the axillary level was applied to allow shoulder exercise. Rotation of the fragments within the plaster caused absorption of callus, and two months later non-union was threatening (Fig. 65). It was avoided only by reinstituting complete immobility for four months longer.

complete immobility of the fragments, consolidation was not complete even after five months It was then believed that the fracture was firm enough to begin shoulder exercises, and an unpadded plaster cast was applied from the wrist to just below the shoulder joint Within two months, the effect of rotation strain was obvious the union broke down, the callus underwent resorption; and established non-union threatened in the tendency to sclerosis of the fractured surfaces. Shoulder movements were stopped and complete immobilisation was reinstituted by a plaster spica; then, after several months of further fixation, consolidation was completed

A plaster cast from the wrist to the axilla in the treatment of indolent fractures of the humerus is comparable to a below-elbow plaster cast in the

treatment of indolent fractures of the ulna. In each case the half-plaster is more dangerous than no plaster at all [1]

Rotation strains in fractures of the shaft of the tibia—Fractures of the shaft of the tibia show the same response to rotation strain The fracture is not immobilised unless both knee and ankle joints are immobilised. A plaster to the knee may control general alignment, but it does not immobilise the fracture, because twisting of the knee can rotate the upper fragment of the tibia within the plaster. The plaster can be twisted round the leg. Close moulding round the foot and ankle gives a secure grip of the distal fragment of the tibia, and every twisting movement of the plaster is transmitted directly to the site of fracture The position is exactly similar to the use of a short forearm plaster for fractures of the ulna, or a wrist-to-axilla plaster for fractures of the humerus *Half a plaster is more dangerous than no plaster* The below-knee cast has no place in the treatment of tibial fractures even in the later stages ; it gives a false sense of security. Either the fracture is already united and the plaster is unnecessary, or the fracture is not united and the plaster is harmful. The plaster should extend from the foot to just below the groin Moreover, the knee joint must be slightly flexed Even a full-length cast does not immobilise a fracture of the tibia completely if the knee joint is extended fully. The terminal degrees of extension are accompanied by a locking movement which binds the femur and tibia and makes them rotate together Rotation of the femur twists the upper fragment of the tibia, and this torsional strain may prevent union. When the fracture is in the upper shaft of the tibia it may even be advisable to include the hip joint in a plaster spica, so that there is no possibility of rotation of the femur /

Rotation and shearing strain in fractures of the scaphoid—It is not many years since a world-famous radiologist said that he had never seen a fracture of the scaphoid unite by bone. This was the day when the application of a cock-up splint for a few weeks was the accepted treatment (Fig 53). Such a splint does not protect the carpus from shearing strain The scaphoid lies half in the proximal row and half in the distal row of the carpus (Figs 67-68) With lateral movements of the joint, the distal fragment tends to move with the distal carpal bones and the proximal fragment with the proximal bones This shearing strain must be controlled by a plaster cast extending round the sides of the wrist and hand, thus preventing adduction and abduction movement.

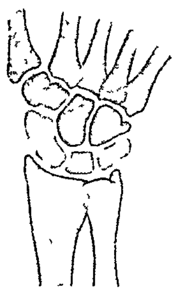


FIG 67

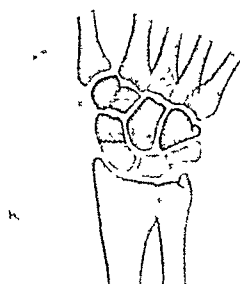


FIG 68

A fracture of the waist of the scaphoid coincides with the line of the mid-carpal joint, and even slight wrist movement causes shearing of the fragments

Is shearing the only strain that is responsible for the frequency of non-union in fractures of the scaphoid ? In the other sites we have discussed, namely, the shaft of the ulna, the shaft of the humerus and the

[1] The " hanging cast " technique advocated by Caldwell and other American surgeons (J A Caldwell, " Treatment of Fracture of Shaft of Humerus by Hanging Cast," *Surg Gynec Obstet* , 1940, **70**, 421) is based on the same principle as the simple gutter splints and collar-and-cuff sling shown in Fig 61, but plaster is used from the wrist to the axilla as described in the case quoted above While adequate for all fractures of the shaft of the humerus in which repair is vigorous and rapid, this technique is obviously dangerous for indolent fractures and will cause non-union In such cases the shoulder joint *must* be immobilised as well as the elbow

shaft of the tibia, it was rotational strain that was responsible Analysis of wrist movement shows that imperfect immobilisation allows similar rotational strain in fractures of the carpal scaphoid bone Dorsiflexion and palmar-flexion are not simple up-and-down movements of the hand. The shape of the articular surfaces shows that this movement consists of a screwing of the proximal row of the carpus into the radius, and of the distal row of the carpus into the proximal row. Examine your own wrist, first at rest and then gripped in dorsiflexion. Dorsiflexion is accompanied by supination of the carpus and hand on the forearm bones; this screwing movement locks the dorsiflexed wrist in the position of strength When the scaphoid is fractured, one thread of the screw is stripped The slightest dorsiflexion or palmar-flexion movement screws the fragments on each other, causes rotational strain, tears the young granulation tissue, produces traumatic hyperæmia and bone resorption, and causes delayed union. It is obvious that a plaster cast with padding is inadequate. Every degree of movement is harmful, and closely fitting unpadded plaster casts are necessary. If this immobility is maintained uninterruptedly all recent fractures of the scaphoid unite by bone, and since in most cases the blood supply has not been damaged, union is secured in eight or more weeks. If the plaster is allowed to become loose, to crack, or to become wet and sodden, and is not immediately replaced, union is always delayed, the fracture may then unite only after twelve or more weeks. If the plaster is occasionally removed for clinical and radiographic examination, and the wrist is left unprotected for several hours or days, union is still more delayed; and if these interruptions are repeated often enough, delayed union passes finally into established non-union.

Shearing and rotation strains in fractures of the femoral neck—Until recent years fractures of the neck of the femur were treated in plaster and



Fig 69
Fracture of the neck of the femur treated in plaster Non-union is due to shearing and rotation movements at the site of fracture



Fig 70
Fracture of the neck of the femur treated by nailing. The fracture has united because immobility is absolute

non-union occurred in about 50 per cent. of cases The modern treatment is to fix the fragments by means of a three-flanged nail, and if there is no complication nearly all of them unite What is the difference between the fixation of plaster and that of a three-flanged nail? In a plaster spica slight twisting and turning of the pelvis, and therefore of the loose femoral head,

is still possible within the plaster There is continued shearing and rotation movement of the fragments. On the other hand, a three-flanged nail affords complete fixation The length of the nail prevents shearing, and the flanges prevent rotation of the fragments. If the nail is perfectly placed, so that there is absolute immobility of the fragments, union is possible in almost every case

In the treatment of fractures of the neck of the femur it is customary to distinguish two types (i) "adduction fractures," in which the shaft is adducted in relation to the femoral head, the fracture being in a relatively vertical plane and without impaction, (ii) "abduction fractures," in which the shaft is abducted in relation to the femoral head, the fracture being in



FIG 71

Adduction fracture neck of femur

The axis of movement of the fragments due to muscle retraction and weight-bearing passes across the plane of fracture There is shearing strain The fracture does not unite unless perfectly immobilised.



FIG. 72

Abduction fracture neck of femur

The axis of movement of the fragments due to muscle retraction and weight-bearing passes through the plane of fracture There is no shearing strain The fracture unites even without treatment

a relatively horizontal plane and with impaction The adduction type never unites unless very special care is taken to secure rigid immobility, preferably by means of a three-flanged nail or some other internal fixation, whereas the abduction type nearly always unites without the aid of a nail, without a plaster spica, and indeed without any treatment at all The difference lies in the fact that when there is adduction displacement the line of weight-bearing and of muscle contraction is such that there is shearing movement between the fragments, whereas when there is abduction displacement the thrust of weight-bearing and of muscle contraction impacts the fragments into each other (Figs 71-72) The adduction fracture has no inherent immobility and fails to unite unless immobilised by the surgeon; but the abduction fracture, by reason of the shape and position of the fragments, is already immobilised

It is true that recent studies have suggested that there are not two different types of fracture, one produced by adduction strain and the other by abduction strain[1]: the "types" are no more than stages of displacement in response to the same injury in which there is twisting of the femoral head on the neck. The first degree of displacement gives the appearance of abduction and impaction; but if the twisting force continues, and the fracture is completed, impaction is broken down so that adduction displacement develops and the plane of fracture appears to change from the horizontal to the vertical. The fact still remains that if the position of the fragments is such that there is shearing between them, the fracture



FIG 73

Two years' old adduction fracture of the neck of the femur treated in a plaster spica. Union is still unsound because there is continued shearing stress



FIG 74

Shaft of the femur has been abducted by a trochanteric osteotomy. The shearing stress has become an impacting force. Union is now soundly consolidated

will not unite, whereas if the position of the fragments is such that there is inherent immobility, the fracture will unite.

This is confirmed by the success that may be achieved by performing an osteotomy below the level of a complete fracture with adduction displacement. If the femoral shaft is abducted at the site of osteotomy, so that the shearing stress is changed to an impacting force, the fracture unites if it is protected only by a plaster spica (Figs 73-74). Such osteotomy is of value not only in the treatment of delayed union and non-union, but is sometimes claimed to be better than the use of nails and wires even in recent fractures.[2]

Shearing and rotation strains after the plating of fractures—It is often said that slow union or non-union is caused by metal plates of the Lane type.

[1] Per Linton. "Types of Displacement in Fractures of the Femoral Neck and Observations on Impaction of Fractures" *J Bone Joint Surg*, 1949, 31-B, 184.
[2] McMurray, T. P. *J Bone Joint Surg*, 1936, 18, 319.

because a slight gap between the fragments is maintained by the rigidity of the plate and continued impaction of the fragments is thus prevented. Although this view is held so widely—indeed almost universally—I am sure that it is wrong. Continued impaction is not essential; slotted plates are quite unnecessary (see Chapter XI). Figure 80 illustrates one of many examples which prove that with adequate immobilisation there is little difficulty in bridging a gap of as much as four or five inches. Why then should there be difficulty in bridging a gap of one or two millimetres? The fact is that there is no such difficulty.

Paradoxical as it may seem, the non-union so often associated with bone plating is again due to inadequate immobilisation. At the time of operation the fragments are immobilised perfectly by the metal plate and screws—so firmly indeed that the limb may be moved in every direction with apparent safety. The surgeon is therefore tempted to discard external splints altogether, or to employ inadequate external splinting, or to use external splints for an inadequate period. But complete and prolonged external splinting is indispensable because the fixation secured by the plate is not sustained. Bone reacts to abnormal pressure by resorption, and the more tightly the screws have been driven in, the more likely it is that they will loosen. If external support is inadequate the pressure is increased and every screw subjected to strain becomes loose. Furthermore, the screws may loosen from toxic reaction in the bone, or from electrolysis. Within a few weeks the fracture is dependent on external splinting for its immobilisation, and rotation and shearing strains lead to the usual, indeed inevitable, sequel of non-union. If plates and screws are to be used it is essential that they should be protected from every strain. Complete immobilisation by full-length plaster casts or splints must be continued until union is sound.

DELAYED UNION FROM INFECTION

The belief that infection of bone is a cause of non-union of fractures has survived for many years. Even recently two American surgeons, reporting non-union in 25 per cent. of infected fractures, displayed no surprise or regret and clearly believed that failure in one fracture out of four had been inevitable. At the same time a British surgeon declared that immediate guillotine amputation was often justified on the grounds that the infected fracture would probably fail to unite, sinuses would continue to discharge, and, sooner or later, a useless limb would be amputated after many years of suffering. What a memory of former days! The fact is that infection is not a cause of non-union; it is a cause only of delayed union. If non-union is allowed to occur, it is due not to infection but to inadequate immobilisation permitted by reason of infection. Immobilisation may be inadequate from the beginning because priority is given to treatment of the infection; complete and uninterrupted immobilisation is sacrificed in order to permit regular inspection of the wound, frequent dressings, and antiseptic irrigations. Alternatively immobilisation may not be continued long enough. A time limit is fixed, and if the fracture has not united within that period it is assumed that the fracture is not going to unite; the label "un-united" is applied at the dictates of a calendar and complete immobilisation is abandoned in favour of a walking splint.

Importance of prolonged and uninterrupted immobilisation—An infected fracture is exactly like a simple fracture except that each stage of repair is prolonged. Bone is destroyed not only by injury but also by infection; bone is resorbed not only by traumatic hyperæmia but also by infective hyperæmia, a gap may have to be bridged. Moreover, whereas simple traumatic hyperæmia subsides within a few days, infective hyperæmia may persist for many weeks or months. Not until infection is healed and



Fig 75

A six months' old compound fracture of femur with delayed union There is sequestration, discharge from sinuses and active infection.

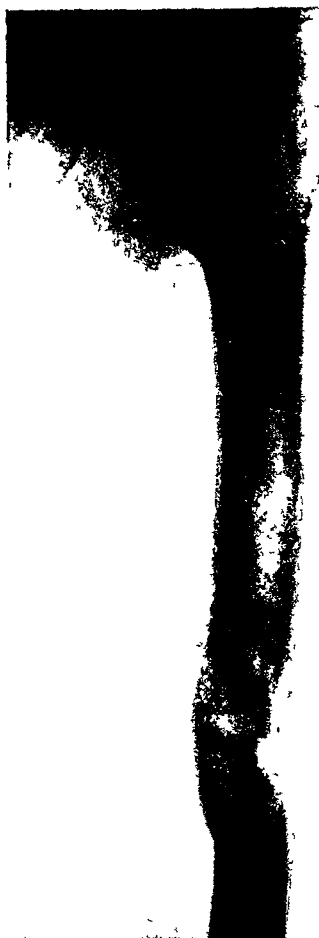


Fig. 76

Because the infection was active, non-union was not established After simple sequestrectomy and immobilisation for several months, the fracture united

hyperæmia subsides can reossification begin, and it may be several months before the infected fracture reaches the stage of repair at which the simple fracture arrives within a few weeks. Throughout these months the infected fracture shows the same susceptibility to shearing and rotation strains Movement tears granulation tissue and creates a plane of cleavage On the other hand, if immobilisation is complete and uninterrupted, and if it is continued not only until infection is healed but for several months afterwards, until losses are made good, infected fractures unite with the same certainty as fractures without infection.

Closed plaster, Winnett Orr technique—The Winnett Orr closed plaster "no dressings" technique is now of little more than historic interest in the treatment of infected fractures, but important lessons were learned from it Free incisions were made, soft tissues were laid widely open so that no deep pockets remained, and the bone was thus drained The wound was lightly packed with vaseline gauze and the skin over which pus would flow was similarly protected with vaseline The fracture was then treated exactly as if it had been a closed fracture by manipulation, traction and immobilisation The plaster cast was changed only at one or two monthly intervals At the time of change of plaster care was taken to permit no movement of the fragments The infected wound healed by granulation at the cost of discomfort, irritation of skin, dermatitis and the revolting smell of retained discharges Very often the wound healed slowly But the important fact was that uninterrupted immobilisation permitted union of the fracture despite infection Complete, continued and uninterrupted immobilisation was possible, and surgeons learned that infection was not in itself a cause of non-union The closed plaster treatment is no longer necessary because nearly all bone infections can now be controlled by penicillin and other antibiotics, and closure of the wound by secondary suture or skin grafting is usually possible within a few days But the principle must be remembered Even if there is persistent infection, union of fractures can be relied upon if immobility is complete, continuous and uninterrupted.



FIG 77 FIG 78

In consequence of removing the whole thickness of the shaft of the tibia at the time of primary wound excision (Fig. 77) the periosteal tube almost completely collapsed so that no more than a slender bridge of bone could be laid down (Fig 78)

Importance of early sequestrectomy—Throughout the stage of active infection radiographs show the characteristic features of delayed union There is bone resorption and porosis, and the fracture has the appearance of a "cavity" with a woolly and ill-defined margin It matters not whether the fracture is six, twelve or eighteen months old, it is not an un-united fracture, it is a fracture with delayed union which will unite if immobilisation is continued The presence of a sequestrum in the region of the infected fracture prolongs the stage of delayed union by causing continued hyperæmia, and the sooner sequestrated fragments are removed the better (Figs 75-76).

The persistence of a sinus is evidence in itself of the presence of a sequestrum or foreign body Even when radiographs fail to disclose a sequestrum the sinus should be explored, fragments of wood, leather or clothing, which are not opaque to X-rays, cause the same reaction and the same delay as fragments of dead bone which are opaque It is surprising how rapidly a fracture will begin to unite after many months of indolence when a discharging sinus is healed by removing the sequestrum that was responsible. The operation does not, however, consist in the scraping of sinuses or the scraping of bone. Rough scraping may remove some sequestra but it produces many more by exposing fresh areas to infection. If it is possible, the sequestrum should be removed by simple dissection with scalpel and forceps, no chisel gouge or surgical spoon being employed In these circumstances the wound often heals by first intention and if the operation is followed by complete and continuous immobilisation the fracture unites soundly within a few months

Exception to the rule of early sequestrectomy—There is one exception to the rule that dead bone should be removed at the first possible moment and as soon as it has separated that is, the sequestrum that consists of the whole thickness of the shaft of a long bone Fragments of the shafts of the femur or tibia, several inches long, may become separated from all soft tissue attachments and undergo avascular necrosis and sequestration If these large fragments are removed at the time of the original wound excision, shortly after injury, or within the first few weeks, the surrounding tube of periosteum may collapse and the subperiosteal hæmatoma be obliterated (Figs. 77-78) There is no longer a continuous hæmatoma between the fragments, and the fracture cannot therefore unite The position is exactly comparable to that which occurs after diaphysectomy for acute osteomyelitis, an operation now discredited because regeneration of the bone so often failed In such a case it is better to defer sequestrectomy for two or three months, until a surrounding





FIG 79 FIG. 80

Compound fracture of the femur in which the proximal fragment was driven out through the skin and buried in a ploughed field Four inches of the shaft sequestrated (Fig 79) but complete regeneration was secured (Fig 80) because sequestrectomy was deferred until subperiosteal ossification assured continuity of the periosteal tube

involucrum of subperiosteal bone has been laid down, thereby ensuring continuity of the shaft (Figs 79-80) When several inches of the shaft of a bone must be reossified, the imperative need for uninterrupted immobilisation is very obvious A single careless movement during the changing of a plaster may refracture the young callus and cause delay, and repeated movements or continued strain prevent the fracture from uniting at all

Non-union of infected fractures—If sequestra have already been removed and the wound is healed, but the fragments have still not been immobilised, delayed union passes gradually into non-union The bone-ends are even more densely sclerosed, and the intervening scar tissue even more lacking in blood supply, than with closed fractures Immobilisation alone is then valueless The sclerosis must first be broken down by operation, and a new traumatic hyperæmia must initiate once more the growth of granulation tissue



FIG 81

Recurrent infection after bone grafting.

A whole-thickness graft including compact bone was wrongly used Moreover the surgeon immobilised the limb only for four months As might be expected the graft sequestrated, non-union recurred, and the operation was wasted

The treatment of old infected un-united fractures was dominated hitherto by the fear of latent and recurrent infection It was suggested that bone grafting should be deferred for twelve months, or even two years, after quiescence of infection Drilling the fragments instead of bone grafting was practised A preliminary manipulation was sometimes advocated in order to determine whether infection could be lighted up A two-stage operation was often recommended—first freshening the bones and cutting the bed and then, two or three weeks later, completing the graft if there had been no flare of infection, presumably if there had been a flare the project was abandoned altogether. When we recall that the source of non-union is not infection but inadequate immobilisation, the situation loses most of its terrors Recurrence of infection is unfortunate because it delays recovery, but beyond this little harm is done Infective hyperæmia supplements the traumatic hyperæmia of operation, and sclerosed bone is resorbed and scar tissue revascularised with even greater certainty, and a successful result may still be expected if immobilisation is prolonged Precautions should certainly be taken to minimise infection An interval of two or three months after complete quiescence and healing of the wound will suffice To wait longer is futile because living organisms may remain locked in the dense bone and scar tissue for the rest of the patient's life No matter how long operation is deferred the risk cannot be excluded altogether It is wise, therefore, when there is a possibility of latent infection, not only to give penicillin or other antibiotics before operation and for a week or two after but also to use only cancellous chip grafts which, unlike grafts of cortical bone, seldom sequestrate even if infection recurs Moreover, special care should be taken to avoid fixation by metal screws and plates, which serve as foreign bodies causing persistent infection and sinus formation

NON-UNION FROM INTERPOSITION OF SOFT PARTS

The first paragraph of this chapter concluded: "There is only one cause of non-union of fractures with a continuous hæmatoma between the fragments —*the cause of non-union is inadequate immobilisation*." It is obvious, however, that if there is no continuous hæmatoma and no ossifiable medium between the fragments union cannot take place no matter how long the fracture is immobilised. Diaphysectomy and the removal of large fragments of the shaft of a bone have already been discussed. collapse of the periosteal tube with complete obliteration of the fracture-hæmatoma prevents regeneration of bone and bridging of the gap Similarly, if a small fragment is avulsed from a bone, traction of muscles attached to the fragment may cause



FIG. 82 FIG 83 FIG 84 FIG. 85

Fractures of the ulnar styloid, patella, internal epicondyle of humerus and tibial tubercle. When a small fragment is avulsed, the fracture-hæmatoma may be obliterated by the interposition of soft tissues, and the fracture fails to unite

attenuation and obliteration of the hæmatoma, as, for example, in avulsion of the epicondyles of the humerus by the flexor or extensor muscles, the tip of the olecranon by the triceps, the great tuberosity of the humerus by the supraspinatus, the tip of a dorsal spinous process by the scapular muscles, a lumbar transverse process by the erector spinæ, the anterior iliac spines by the sartorius or rectus femoris, or the tibial tubercle by the quadriceps

In other fractures the hæmatoma may be obliterated by an interposed flap of living tissue which seals the fractured surface of one fragment In fractures of the patella with separation, the surface of the proximal fragment is covered by the aponeurosis of the quadriceps expansion In fractures of the base of the medial malleolus, bony union may be prevented by a flap of periosteum torn from the adjacent surface of the tibia and interposed across the line of fracture Occasionally in a fracture of the middle third of the shaft of the radius, the surface of one fragment may be sealed by fibres of the pronator teres insertion In fractures of the upper shaft of the femur, sustained as the result of considerable violence, one fragment may be driven so forcibly through surrounding muscles that, despite traction and manipulation, the fractured surfaces cannot be brought together, and the interposed mass of living muscle prevents union In all such fractures operative treatment to restore continuity of the bone and apposition of the

fractured surfaces is a necessary prelude to the immobilisation by which union is secured

Summary of the causes of Slow union, Delayed union and Non-union

Slow union with indolence—

Impaired blood supply
Excessive traction

Delayed union with cavitation—

Inadequate immobilisation
Infection

Non-union with sclerosis—

Diaphysectomy
Interposition of soft tissues

Slow union and Delayed union, with too early cessation of immobilisation cause Non-union

ESTABLISHED NON-UNION OF FRACTURES

When non-union of a fracture is established, and there is dense sclerosis of the fractured surfaces with a layer of mature fibrous tissue between the bone-ends, or even the development of a false joint, the essential treatment is to cut through sclerosed bone and fibrous tissue, re-freshen the fragments, and immobilise the bones continuously and without interruption for a long enough period Union failed on the first occasion because immobilisation was inadequate, and the main purpose of operative treatment is to go back to the beginning, restore the conditions of a recent fracture, and make sure that immobilisation is indeed adequate Some surgeons have been content to re-freshen the fragments by breaking them up with a chisel, osteotome or drill, and then rely on the external fixation of splints or plaster But since the fracture must in any event be exposed it seems wiser to combine a re-freshening operation with bone transplantation.

There has been much debate as to whether transplanted bone dies or survives, and whether transplanted bone cells are capable of promoting osteogenesis These problems will be discussed in Chapter XIV Meanwhile it must be recognised that even grafts of cadaveric bone have succeeded, and that autogenous grafts which accidentally fell to the floor of the operating theatre and were boiled before being transplanted have also succeeded Whatever merit transplanted bone may have in promoting osteogenesis, bone grafting operations succeed on purely mechanical grounds for two reasons (1) in the preparation of a bed for the graft, sclerosed bone and fibrous tissue is effectively cut through, so that the conditions of a recent fracture are reconstituted, (2) the inlaying or onlaying of a graft, even if it is dead, provides an internal splint which augments the necessary fixation of bone fragments and provides a scaffolding for the laying down of new bone

CHAPTER III

ADHESIONS AND JOINT STIFFNESS

In the last chapter emphasis was laid on one fundamental principle of treatment—a principle that applies to every type of fracture whether complicated or simple, infected or not infected, recent or old, treated by manipulation or by operation—

Every fracture must be treated by complete and continuous immobilisation until union is sound

This complete fixation of the fractured bone involves immobilisation of the joints above and below the fracture There is a second principle of equal importance which must also be in the mind of the surgeon from the beginning of treatment—

Every joint which does not need to be immobilised must be exercised actively from the first day of injury.

Failure to obey the first law of treatment is a source of delayed recovery and may be responsible for non-union Failure to obey the second law is a no less frequent source of delayed recovery and may be responsible for complications even more serious because they often remain permanently despite treatment

Stiff fingers due to excessive splinting—If a fracture of the wrist is immobilised in splints which extend over the finger joints (Figs 86-87), the



Fig 86



Fig. 87

"*Rigid splints mean rigid fingers*"—Sterling Bunnell

The bar of the wooden splint and the plaster in the palm prevent flexion exercises of the fingers This immobility is unnecessary and usually leads to disastrous results

fingers become so stiff that weeks and months of treatment are necessary to mobilise them once more They may never recover, rigidity may remain as a permanent incapacity The gravity of the disaster cannot be over-emphasised Permanent stiffness of the fingers means permanent crippling The whole purpose of the limb is lost when the prehensile function of the fingers is lost Of what avail is perfect union of the fracture if the hand is crippled? If the patient is a working man he may never work again

(Fig 88) But there was no necessity to immobilise the fingers at all. The joints were not stiff at the time of injury, they became stiff in consequence of the treatment The treatment itself caused months of delay and even permanent crippling which need never have arisen The patient would have done better to have stayed at home Even without any treatment the result could not have been worse. So disastrous has been the result of excessive immobilisation and neglect of joint exercises that Lucas Championnière and, in recent years, Perkins[1] and others determined to do away with splints altogether. Even delayed union and mal-union were not so bad as irrecoverable joint stiffness. The limbs were simply supported in a sling and massage and joint exercises were practised daily



Fig 88

Simple wrist fracture twelve months after treatment as in Figure 87. The patient is trying to make a fist, but the hand is so crippled that he will never work again

Preventing stiffness despite complete fixation of fracture—There is, in fact, no difficulty in combining the two principles—immobilisation of the fracture, and mobilisation of the joints The wrist fracture is completely immobilised in a closely fitting plaster extending from the back of the metacarpal heads to just below the elbow with a single width of strapping or bandage in the palm so that there is no restriction of flexion movement of the fingers From the first day, full bending and straightening movement of every finger joint is practised. The discomfort that may at first be experienced soon passes off, and the exercises must be continued for not less than five minutes every hour of the day (Figs 89-90) The elbow must also be flexed fully and extended fully, and the limb be raised to the side of the head and rotated in each direction so that the shoulder does not become stiff If plaster immobilisation is complete, the patient is encouraged to use the limb for dressing, eating, and light household duties (Figs 92-93) It may even be possible after some weeks for men to go back to work When, finally, the plaster is removed, every joint of the limb has normal mobility except the wrist itself Moreover, the muscles are well developed, wasting has been minimised, and disuse porosis of bones has been prevented Perhaps the most important point is that the limb has never been disconnected from the brain, the patient has not forgotten how to use it, he is not terrified at the prospect of using it He has been so busy practising exercises that he has had no time to sit back lamenting his fate and developing functional complications.

The surgeon must first see that no splint, plaster, strapping or bandage interferes with finger movement He must not use any position of immobilisation that makes finger exercise impossible For example, the position of right-angled flexion of the wrist used in the Cotton-Loder

[1] Perkins, G "Fractures" *Oxford Med. Pub*, 1940

treatment of wrist fractures makes it impossible to flex the fingers. The position is both unnecessary and dangerous.

The surgeon's duty is still not done Not only must there be no obstacle



FIGS 89-90

Perfect immobilisation of the fracture need not prevent finger movements The joints are exercised hourly throughout the day.



FIG 91

The shoulder must not be allowed to stiffen The arm is put over the head and behind the back many times a day



FIG 92



FIG. 93

In many cases household activities and light work can be continued throughout the period of immobility of the fracture

to movement, the patient must actually practise the movements that are now possible and he will not do this on his own initiative His instinct is to guard against the slightest movement of any joint When he has been told to exercise he still doubts, or is afraid, or is unwilling to accept the initial discomforts of finger exercise Every day of postponement increases

the difficulty The patient must be seen daily and be encouraged, cajoled, stimulated and when necessary bullied into activity. Every joint must be flexed fully, the interphalangeal joints by flexing the fingers tightly into the palm, and the metacarpo-phalangeal joints by reaching with the finger tips to the front of the wrist The index finger must not escape attention (Fig 95) Flexion of this digit may be difficult owing to the position of the thumb, and stiffness confined to this finger is very often seen The patient must be taught to keep the thumb out of the way while he bends the fingers.

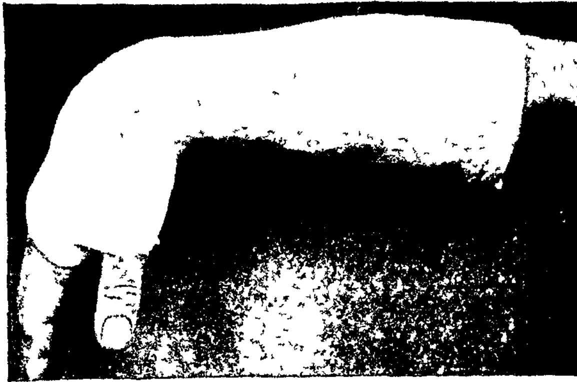


FIG. 94

If the wrist is flexed to the right angle it is impossible to bend the fingers Even though the fingers are not actually splinted, serious stiffness will develop.

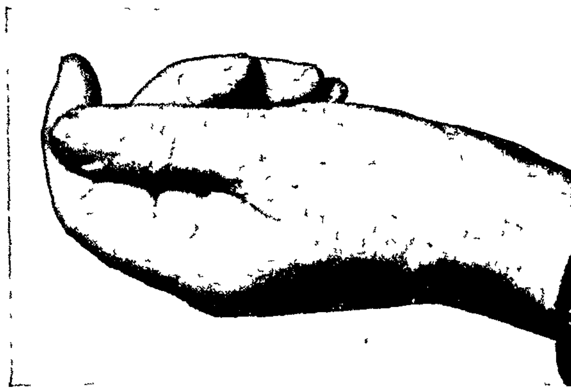


FIG 95

The position of the thumb is obstructing flexion movement of the index finger. This is a common cause of stiffness of this finger.

When the fracture does not involve the wrist but is at a higher level, so that immobilisation of the elbow and shoulder joints is necessary, finger exercises are even more imperative When the plaster is discarded, the patient whose hand is normal has an incentive to use the limb and the stiffness of the proximal joints recovers If the hand is stiff and useless the patient has no incentive He regards the limb as crippled It never occurs to him to use it There is then great difficulty in persuading him to undertake the exercise necessary for recovery.

Finger exercises prevent stiffness even of immobilised joints—We have not yet told the whole story, finger exercise is more than an end in itself It does much more than prevent stiffness of the fingers, it actually prevents stiffness of the wrist joint which has been immobile throughout. If two wrist joints with identical injury are immobilised for the same period, one in splints over the fingers which cause functional disuse, and one in a dorsal plaster which leaves the fingers free and permits functional activity, although both wrists are immobilised equally the first becomes very much stiffer than the second In other words, it is not immobility alone that causes joint stiffness, it is immobility *and* functional disuse A fracture of the scaphoid bone with delayed union may require immobilisation for six months, or even longer, and it might be assumed that this would cause serious stiffness of the wrist. This would be true if there was complete functional disuse as well as immobility But where function is maintained by finger exercises involving contraction and relaxation of the forearm muscles, even although

the wrist itself is not moved, the stiffness is minimised Immediately the plaster is removed the range of movement is more than half of normal (Figs. 96-97), and within a few weeks, by the practice of active exercise, it is normal.

Increase in movement while a joint is immobilised—The range of movement of a joint may even increase while it is immobilised in plaster A fractured wrist taken out of plaster at the third week is very stiff If it



Figs 96-97

Movement of the wrist immediately it is removed from plaster after six months' immobilisation for delayed union of a fractured scaphoid The functional activity of finger exercise has prevented stiffness of the wrist

had been left in plaster for two or three weeks longer it would have been less stiff, and if immobilisation is needed for several months it continues to gain movement, provided only that functional activity is maintained by finger exercise Old united fractures of the ankle with severe stiffness are often best treated by immobilisation in a walking plaster. If functional activity is continued, the range of movement increases gradually and it may recover almost to normal during the two or three months that the joint is immobilised

CAUSES OF ADHESIONS AND JOINT STIFFNESS

What is the cause of this stiffness which is the consequence not of immobility but of functional inactivity?[1] It arises from the adhesion of capsular plications and synovial reflections, and the "glue" of which the adhesions are made is the fibrin of sero-fibrinous exudates. Any persistent or recurrent sero-fibrinous exudation may cause adhesions. Immobility of a joint cannot in itself give rise to sero-fibrinous exudation, but muscular inactivity is responsible for venous and lymphatic stasis; the circulation is sluggish and the tissues become waterlogged. Here is the source of sero-fibrinous fluid which produces capsular and intermuscular adhesions. The causes of joint adhesions after injury may be enumerated:

1. Functional inactivity and disuse—circulatory or lymphatic stasis and waterlogging of the tissues with sero-fibrinous fluid
2. Joint injury—traumatic sero-fibrinous exudation from torn capsule or a neighbouring fracture, especially if the injured joint is forcibly moved in the early stages
3. Recurrent œdema—traumatic œdema and recurrent gravitational œdema especially in the lower limbs
4. Infection near the joint—inflammatory sero-fibrinous exudation spreading from a neighbouring focus of infection
5. Foreign bodies, especially skeletal traction pins close to joints with low-grade inflammatory exudation spreading from the pin track.
6. Repeated passive stretching and forcible manipulation of a stiffened joint—traumatic sero-fibrinous exudation from the torn adhesions

Functional inactivity and disuse—Figures 96 and 97 show a joint which, though immobilised for six months, had been functionally active throughout that time and little or no stiffness developed. Figures 98-100 show the converse. This joint had never been completely immobilised but there was serious functional inactivity, and even eighteen months later the joint was almost completely stiff. The injury was a simple fracture of the malleolus associated with swelling and ecchymosis. The joint was strapped. The patient lay in bed for three weeks and then tried to walk. Meanwhile the foot had dropped. The heel could not be put to the ground. The patient could put no weight on the limb. Weeks went by but functional activity was still impossible. A year later the victim could walk round the kitchen with crutches but no more. The foot and ankle were almost completely stiff. The practitioner labelled it "arthritis". Others thought that it was a trophic change of neuro-vascular origin. Under anæsthesia the adhesions were stretched so that the foot could be raised to the right angle and a plaster was applied in this position. With the painful joints protected, and the heel and sole flat on the ground, walking became possible for the first time. Within two weeks the patient walked five miles a day. Within two months the plaster was removed and foot and ankle movement had recovered to more than half of normal. In four months recovery was complete. Adhesions round joints which had never been immobilised had been cured by plaster immobilisation with functional activity.

[1] Watson-Jones, R. "Adhesions of Joints and Injury." *Brit. med. J.*, 1936, 1, 925

Joint injury—A simple joint injury which is not complicated by functional disuse or other sources of repeated exudation does not cause serious or lasting stiffness Even traumatic dislocation of the knee or shoulder joints, in which there is extensive capsular injury, causes no more than temporary stiffness. But if the injured joint is treated by early movements, and the



Figs 98, 99, 100

Eighteen months after simple malleolar fracture of the ankle. Although the joint was never immobilised, functional inactivity has caused adhesion formation The joint is in so much equinus that the patient cannot walk. The ankle will gain movement while it is immobilised in a walking plaster.

torn tissues are not protected, there is *repeated* exudation, and it is the soaking of tissues with fibrinous serum day after day that causes dense adhesions The practice of treating dislocations and fractures of the shoulder by immediate movement defeats its own object, instead of preventing stiffness it aggravates stiffness The correct treatment is to protect and support the torn tissues until they are healed, to continue exercises of the distal joints, and to begin active but not passive movements of the injured joint after two or three weeks

Reactionary traumatic œdema—Fractures of the lower forearm are sometimes associated with severe swelling of the fingers The fingers themselves are uninjured but œdema spreads from the site of injury especially if the forearm and hand are encased in plaster The natural instinct of the alarmed patient is to avoid any movement Often the practitioner shares the patient's fear, he accepts the swelling as an index of the severity of injury and prescribes rest until it has subsided Exactly the opposite treatment is urgently indicated Œdema is "glue" Adhesions are forming hourly, and the more swollen the fingers the more imperative is active exercise Exercise will not only prevent the adhesions from becoming more dense but will accelerate the venous return and help the œdema to subside The fracture must be reduced and immobilised at once so that exercise can be practised painlessly. The patient should be recumbent for a day or two with the limb elevated between pillows in order to reverse the effect of gravity. It is in this type of case that the most severe and intractable stiffness of the fingers may develop. Prompt action is essential.



Fig 101

Severe œdema of the leg persisting two years after fracture of the tibia and causing serious stiffness of the ankle This could have been prevented by elastic support applied immediately after removal of the plaster.

Gravitational œdema—When a leg or ankle fracture has been immobilised in plaster, removal of the rigid external support is followed by œdema of the leg which increases during the day and subsides during the night (Fig 101). This continues until the musculature and circulation of the limb are toned up by active exercise. If recurrent œdema is not controlled by elastic support, the ankle tissues are soaked each day in fibrinous serum and the joint, which is already somewhat stiff, may become steadily stiffer This source of adhesion formation can be prevented by applying an elastic dressing as soon as the plaster is removed, and keeping it in position for a few weeks, until the tendency to œdema subsides

Rigid clawed toes—When a leg or ankle fracture is immobilised in plaster and toe exercises are neglected, the disuse, immobility and recurrent œdema cause stiffness of the toe joints This is particularly serious if the toes have been immobilised in the clawed position The corns which develop on the dorsal surfaces are the least important disability The significant point is that rigidly hyperextended toes are functionally useless, for they cannot be flexed to the ground (Fig 102) Normally, with every step forward, weight is transmitted to the toes, and as they flex at the metatarso-phalangeal joints, weight-bearing beneath the metatarsal heads is reduced Rigid clawing of the toes interferes with this mechanism, and the patient walks on the metatarsal heads themselves There is crippling pain and a sensation

as of "walking on small stones." This is a most frequent source of disability and it may keep men off their work for many months after the fracture itself has recovered It is to be avoided by moulding the plaster to the transverse arch with the central metatarsal heads elevated and all the toes flexed, and by insisting on regular toe exercises especially flexion of the metatarso-phalangeal joints Swelling of the toes should be controlled by periodic elevation of the limb



Fig 102

United fracture of tibia with rigid clawed toes due to disuse and recurrent œdema Walking on the unprotected metatarsal heads causes severe pain and prolonged disability.

Infection near joint—A common example of dense adhesion formation round normal joints, which though uninjured and not infected are near a source of infection, is seen in the septic hand The infection causes sero-fibrinous exudation and there may be œdema of the whole hand Very often the infected finger itself is the least serious element of the ultimate disability; there is board-like rigidity of every joint, and recovery is then exceptional Active exercises of every normal finger from the very onset of injury or infection is imperative The hand must not be enveloped in bandages, only the infected finger should be immobilised The swelling and œdema must be controlled by elevation of the limb If it becomes obvious that the finger is irreparably damaged it should be amputated at once in order to save the rest of the hand

Foreign bodies near joint—Metallic foreign bodies may give rise to irritative changes in adjacent tissues with a low-grade toxic reaction If a metal screw, plate or wire has been introduced into a bone so close to the synovial tissues that the reactionary exudate involves the synovia and capsule intractable stiffness arises from the adhesion formation. Fractures of the olecranon are sometimes treated excellently in all other ways except that wire made of a toxic metal has been used for fixation, there may then be permanent limitation of extension of the joint Figure 103 shows a fracture of the external condyle of the humerus, perfectly reduced by operation, but fixed in position by two screws, the irritant effect of which can be seen in the resorption of bone around them There was permanent stiffness of the joint It is now recognised that whereas some



Fig 103

Metallic foreign bodies produce an irritative sero-fibrinous exudation and, if close to the synovial reflections of a joint, adhesions and limited movement

metals and alloys, such as vitallium and 18/8 stainless steel, are inert in the body, being neither toxic nor electrolytic, others cause an irritant reaction with resorption of adjacent bone and loosening of the metal If such metals are close to the synovial reflections of a joint the irritant reaction also causes stiffness of the joint

Skeletal traction pins—Skeletal traction pins, wires and callipers are foreign bodies still more potent in promoting adhesion formation because they penetrate the skin and, after some weeks, low-grade infection of the pin track occurs. Experienced surgeons may be unconvinced by the case shown in Figure 104 and will maintain that infection of a pin through the calcaneus, so severe as to necessitate guillotine amputation of the leg, should not occur This is true But slight low-grade infection cannot be avoided [1] There may only be a few granulations, a trace of serous discharge, or perhaps slight



Fig 104

A result of skeletal traction

Guillotine amputation following infection of an os calcis pin track

general thickening of the tissues (Fig 105) If the pin track is close to the synovial and capsular reflections of a joint, this degree of inflammatory change will be quite enough to bind down these tissues with strong dense adhesions

Supracondylar skeletal traction—In the supracondylar region of the knee joint a traction pin usually causes adhesions which are far more dense and resistant than the simple adhesions of immobility and disuse Movement beyond the right angle may never be regained (Fig 106) Certainly this does not apply to every case , it depends on the degree of reactionary exudation. The bigger the wound at the site of insertion of the pin the greater the danger The greater the movement of the pin in its track the greater the danger Ice tong callipers are the worst , pins to which the calliper is attached and which therefore rotate in their track are the next worst Kirschner wires are perhaps the least harmful But no form of supracondylar skeletal traction is entirely safe The method has gained a wholly undeserved popularity and has been responsible for many stiffened knee joints which could have retained normal movement

[1] Kennedy, R H "Treatment of Fractures of Shaft of Femur" (including forty-two cases of skeletal traction with twenty-one infections) *Ann Surg*, 1938, 107, 419

Tibial tubercle traction—The tibial tubercle is a relatively safe region because even if slight infection does occur, it is too remote from the periarticular tissues to cause joint adhesions. The objection that tibial traction stretches the knee joint ligaments, whereas supracondylar traction does not, applies only when traction is excessive. If excessive traction is not used, the slight laxity of ligaments which develops after tibial traction is no greater than that which accompanies any form of immobilisation of the knee joint, even immobilisation in a plaster spica without traction It recovers fully when the tone of the thigh muscles is regained

Calcaneus traction is again unsafe, for the reactionary exudation may bind the synovial reflections of the subastragaloid joint and cause permanent limitation of inversion and eversion movement. If continuous traction



FIG 105

Diffuse synovial thickening of the left knee from low-grade infection of supracondylar pin track The slightest infection in the supracondylar region causes intractable stiffness



FIG. 106

Fracture of the shaft of the femur treated by supracondylar traction, three years after injury. There is still only 90° of flexion due to adhesions from low-grade pin track infection

is required for a fracture of the leg bones the pin should be inserted through the lower shaft of the tibia about two inches above the ankle joint

Olecranon traction—This is one of the most unsafe regions The elbow joint is particularly susceptible to adhesion formation, and even slight reactionary exudation close to the capsule of the elbow causes intractable stiffness In my own practice olecranon traction has never been needed I know of no fracture that cannot be treated better by other methods

Metacarpal head traction—A pin should never be left in the metacarpals unless it is incorporated in the plaster cast so securely that it cannot move

There are occasions when skeletal traction is useful and necessary; but pins must be inserted only with a proper respect for the harm that may result The treatment must be reserved for the cases that really need it The modern tendency to drive in pins on the slightest provocation is quite unjustifiable To treat a supracondylar fracture of the humerus or a Pott-Dupuytren fracture of the ankle by skeletal traction is to admit lack of manual dexterity To treat a simple fracture of the femur by inserting four pins, two at the top and two at the bottom, carries mechanics into surgery to an unwarranted degree To treat a perfectly straightforward fracture of the humerus by pins at the upper end and at the lower end, in order to mobilise the shoulder and elbow from the beginning, betrays an imperfect appreciation of the causes of adhesion formation and joint stiffness.

Massage and passive stretching—'Massage and movements" often prescribed as a treatment for joint stiffness, are in fact among the commonest of the causes of joint stiffness When the adhesions already formed are violently stretched or torn there is reactionary exudation which produces fresh adhesions. If this treatment is repeated day after day the traumatic exudation recurs, and although every day some adhesions are stretched, many more develop Such treatment delays the recovery of movement in every joint, and in the more susceptible joints of the upper limb—the finger, elbow and shoulder joints—measurements show that movement steadily decreases so long as the treatment is continued Enthusiastic masseuses are almost incapable of resisting the temptation to force stiff elbow joints They should not be tempted An elbow joint stiff after injury should not be sent to a massage department, it must be treated only by the patient's own active exercises Stiff finger joints are equally susceptible The one certain way of converting temporary stiffness of a finger into permanent stiffness is to stretch the joint In former years one often used to hear of masseuses who soothed the patient's anguish by congratulating him every time the joint cracked Such treatment was entirely indefensible.

Many other forms of passive stretching used to be practised Patients with stiff elbows were encouraged to carry buckets of water, or hang from overhead beams Increased stiffness was inevitable (Figs 107-109) A surgeon can in fact decide whether an elbow is being stretched passively merely by recording its movement A protractor is used and the range is recorded every other week (Fig 110) Movement recovers by 5° or 10° each fortnight If movement is not recovering the possibility of passive stretching must be suspected. If movement is less than it was a fortnight previously, the surgeon *knows* that the joint has been stretched

Manipulation under anæsthesia—It is too generally assumed that if there are adhesions round a joint a manipulation will cure them Manipulation is a two-edged sword The procedure will undoubtedly break down adhesions, but that very process gives rise to fresh exudation from the torn fibres which may produce fresh adhesions The new adhesions cannot be prevented by vigorous and forcible after-treatment for, as we have seen, this produces still further adhesions Indeed the balance between adhesions cured and adhesions produced may be a very delicate one

Adhesions must never be stretched under anæsthesia while they are still young and vascular, nor while the limb is still subject to recurrent œdema Manipulation should be deferred until movement is no longer being regained by the patient's own active exercise, and this stage must not be determined by mere guesswork Joints often recover movement so slowly that the patient himself can judge no difference from week to week. The surgeon certainly cannot recall the exact range unless it has been measured accurately and recorded If week after week the range is identical a manipulation may be considered. When this criterion is insisted upon it will be found that the indication seldom arises

If it is decided to manipulate the procedure must be carried out with full muscular relaxation and the utmost gentleness If the joint is manipulated and there is no audible snap of localised adhesions, but a gradual giving way of a solid mass of diffuse adhesion, the manipulation will do little good and may even aggravate the stiffness. If on the other hand the



Fig 107



Fig 108



Fig 109

The causes of permanent stiffness of the elbow

Passive stretching of any joint defeats its own object and aggravates the stiffness The elbow is particularly susceptible Stretching by a masseuse, by carrying weights or by hanging from overhead beams is indefensible

adhesions are heard to snap and are obviously localised, the prognosis is better, provided that too much is not done at one time It is far better to perform two gentle manipulations, with an interval between of not less than three months, than to perform one severe manipulation which must be followed by a severe reaction. After the manipulation, treatment should be entirely by the patient's own active exercise. All the benefit will be neutralised if it is followed by repeated passive and forcible stretching Finally, if measurements prove that a manipulation has reduced the range of movement of a joint, the manipulation should not be repeated

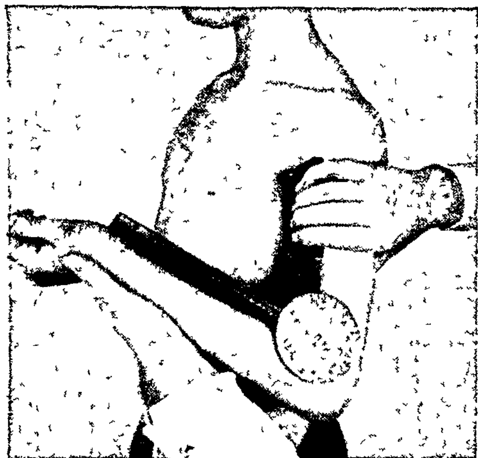


FIG 110

Range of movement must be recorded accurately with a protractor. If it is decreasing, the surgeon knows that passive stretching is being employed

Futility of manipulation of stiff joints—In my own practice the treatment of a stiff joint by manipulation under anæsthesia is now so exceptional that it is regarded as a special event—an unhappy event, implying admission of failure In the last five years, having seen hundreds of joints that were stiff after injury, I have manipulated six knees, two elbows and four shoulders—always as a last resort and never with any certainty of success During the same period I have seen scores of patients whose stiff joints had already been manipulated once or many times without benefit, who were completely cured by no other measure than that of proper supervision of active exercise One had submitted to no less than five manipulations of the shoulder in two years, and it was proposed to operate on his biceps tendon Within three months he was completely cured by active exercise alone It is to be emphasised, however, that the simple prescription of active exercise is almost worthless The last patient I saw with a stiff shoulder had been treated by a surgeon who was well aware of the dangers of passive stretching and repeated manipulation, active exercises had been prescribed, and the patient had attended a physiotherapy department for supervision on alternate days After twelve months he gained only 90° of abduction movement But when he was admitted to hospital, and the very same exercises were practised many, many times throughout the day, under hourly supervision, with constant encouragement and stimulation, he regained almost normal movement within three weeks

Extra-articular causes of joint stiffness—All the causes of joint stiffness that have been discussed so far relate to adhesion formation in the capsule and synovial reflections of a joint But stiffness may also arise in consequence of the fixation of muscles at a distance from the joint In injuries of the thigh, anchorage of the quadriceps muscle is an important cause of stiffness of the knee. This can often be minimised and even prevented by early practice of quadriceps exercises The great importance of maintaining the tone of the quadriceps in order to preserve normal muscle control of the knee joint will be discussed in later chapters, quadriceps muscle insufficiency is itself enough to cause recurrent synovitis of the unguarded

knee joint, especially when there is early arthritis, or injury to the ligaments: whereas even after serious knee joint injuries with rupture of the lateral and cruciate ligaments, recovery may be complete if the quadriceps tone is maintained (Figs 111-112).

It is in open infected fractures of the femur that anchorage of the thigh muscles is most difficult to avoid The more comminuted the fracture, the greater the penetration of muscle by bone fragments, the more prolonged the infection, and the lower in the thigh the site of fracture, the greater is the problem of knee stiffness Preventive treatment is imperative. The essential measures are: first-aid protection of the fracture and continuous immobilisation thereafter in order to prevent muscle injury; prompt excision of wounds and penicillin therapy in order to control infection,



Figs 111-112

Range of movement and muscular development of the knee at conclusion of three months' plaster immobilisation for complete backward dislocation with rupture of the cruciate and lateral ligaments



Fig 113

The quadriceps was never allowed to waste Exercise was practised hourly every day of the three months Two months after removal from plaster the knee is functionally normal despite ligamentous instability

and early quadriceps contraction in order to prevent fixation If the muscles become firmly anchored, manipulation under anæsthesia is usually futile—the usual consequence is rupture of the ligamentum patellæ or fracture of the patella Operative stripping of the muscle from the bone sometimes helps, but as a rule there is no better alternative than lengthening the quadriceps tendon which usually increases the range of flexion movement, perhaps to the right angle, but often at the cost of loss of active control of the last twenty or thirty degrees of extension movement

STIFFNESS OF JOINTS AND SUDECK'S ACUTE BONE ATROPHY

It has been shown that adhesion formation in the capsule and synovial reflection of joints after injury is usually due to œdema, repeated injury, passive stretching, manipulation, infection or the irritation of foreign bodies Such stiffness can nearly always be prevented by active exercise Some joints, however, stiffen more readily than others The joints of adults stiffen more than the joints of children Joints that are the site of arthritis, particularly atrophic arthritis of the rheumatoid type, are specially prone to stiffness in consequence of immobility Even when there is no obvious predisposing cause an occasional patient with a fractured wrist, for

example, may have the utmost difficulty in preventing stiffness of the fingers. Increasing pain is associated with the development of a glossy skin, porotic bones, and spindling of the fingers. In its more severe form it is known as Sudeck's post-traumatic acute bone atrophy.[1-6]

Sudeck's post-traumatic osteoporosis—This condition usually occurs in the fingers and hand, but sometimes in the foot. The pain and loss of function are usually out of all proportion to the degree of injury. It occurs less frequently after fractures than after simple wrenches, strains or crushes, which very often have not been immobilised at all. The hand becomes smooth, glossy, swollen and painful, and it is held immobile by the patient who becomes terrified of any movement. There is marked porosis of the bones. Stiffness of the joints may progress to the stage of contraction of ligaments and even erosion of articular cartilage. The local temperature is increased; oscillometry shows high waves, and plethysmographic studies show an average increase of blood flow of 30 per cent. The morale of the patient is soon impaired and functional disorders arise.



FIG. 114

Porosis of bones in disuse atrophy and Sudeck's acute bone atrophy is first noticed in the terminal phalanges and later in the whole hand

Because the changes in the hand resemble those of causalgia from peripheral nerve injury, and sometimes respond favourably to sympathetic block, it has been postulated that afferent impulses from the region of injury give rise to a "reflex neurovascular sympathetic disorder." It must be recognised, however, that this syndrome differs only in degree from the ordinary disuse change that occurs after every wrist injury when exercises are neglected. If a patient is once allowed to develop stiffness of the fingers there is every likelihood that stiffness will be progressive. The more painful the movement, the less the patient is inclined to exercise—and if he is left to his own devices the more certain it is that stiffness will increase with the usual sequelæ of pain, porosis of bone, glossiness of skin, and spindling of the fingers. Many cases of neglected treatment have been explained away by the convenient diagnosis of Sudeck's atrophy.

[1] Sudeck, P. "Ueber die akute entzundliche Knochenatrophie." *Arch. klin. Chir.*, 1900, **62**, 147.
[2] Leriche, R., and Fontaine, R. "Des osteoporoses douloureuses post-traumatiques." *Pr. Méd.* 1930, **38**, 617.
[3] Middleton, D. S., and Bruce, J. "Post-traumatic Osteodystrophy at Joints." *Tr. Med.-Chir. Soc. Edinb.*, 1933, N.S. **48**, 49.
[4] Gurd, Fraser B. "Sudeck's Atrophy." *Ann. Surg.*, 1934, **99**, 449.
[5] Simpson, B. Soutar. "Post-traumatic Decalcification of the Foot." *J. Bone Joint Surg.*, 1937, **19**, 223.
[6] Oppenheimer, A. "The Swollen Atrophic Hand." *Surg. Gynec. Obstet.*, 1938, **67**, 446.

It is to be noted also that this complication occurs much less often in fractures that are immobilised than in soft tissue injuries that are regarded as trivial and are not protected in such a way as to facilitate active movement of the fingers. It is more than likely that in many cases it would never have occurred if the soft tissue sprain or crush had been protected by means of a plaster slab so that finger exercise could have been practised from the beginning without pain.

In former editions of this book I have written of Sudeck's atrophy: "I doubt very much whether the condition exists, except as the result of neglected disuse changes," and, although it must be admitted that some patients are predisposed to such changes, possibly by reason of hormonal disturbance, I still think that this is basically true.

Treatment—Active exercise must be urged with all the powers of persuasion at the surgeon's command. If there is an unprotected injury of the wrist or hand, a dorsal plaster slab should be applied. If there is œdema, the limb should be elevated. Exercises must be supervised hourly—the patient must not be allowed out of sight. The temptation to stretch and force the joints must be avoided rigidly. If these steps are taken promptly the calamity may often be averted.

Attempts have been made to localise the trigger points of pain and inject them with novocaine; the median and ulnar nerves have been injected with novocaine just above the wrist; but as a rule these efforts have failed. On the other hand, it is worth injecting the upper thoracic sympathetic ganglia with 20 c.c. of 1 or 2 per cent. novocaine. The limb becomes flushed and warm, spasm is relieved, and exercise is facilitated. Pre-ganglionic sympathectomy has also been undertaken. But active exercise remains the sheet-anchor of treatment, and the main purpose of novocaine block and sympathectomy is to facilitate such exercise.

Possible hormonal basis of joint stiffness and Sudeck's atrophy after injury—Recent studies of the "adaptation syndrome"[1] and the hypophysio-adrenal defence to stress may have a bearing on the problems of joint stiffness, Sudeck's atrophy and arthritis after injury. In the adrenal cortex there are hormones, normally in balance, which have opposite effects on the skeletal joints, namely: the inorganic mineralo-corticoids (desoxycorticosterone) which promote fibroplasia and in rats cause arthritis with erosion of cartilage and joint contracture; and the organic gluco-corticoids (cortisone) which resorb fibrous tissue and relieve rheumatoid arthritis. In adaptation to various types of stress—cold, fatigue, fasting, burns, infections—and in response to the stimulus of the adreno-cortico-trophic hormone of the pituitary, the adrenal glands enlarge and there is increased secretion of corticoid hormones by which defence against the stress factor is established. But in certain circumstances there may be disorder of this defence mechanism with, for example, an excessive secretion of mineralo-corticoid hormone and therefore predisposition to stiffness, arthritis and other joint changes. The exact influence of these hormones, and their relationship to ascorbic acid, is still the subject of many researches and at this stage we can only conjecture that there might quite possibly be a hormonal basis for traumatic arthritis and joint stiffness.

[1] Selye, H. "The General Adaptation Syndrome and the Diseases of Adaptation"—a critical survey of the literature. *J. clin. Endocrinol*, 1946, 6, 117.

CHAPTER IV

MYOSITIS OSSIFICANS AND TRAUMATIC OSSIFICATION

We have seen that passive stretching of joints always defeats its own object and that instead of increasing the range of movement it aggravates the stiffness This is not the only disastrous sequel It is often responsible for the formation of masses of new bone round the joint, a complication that has become known as myositis ossificans traumatica The title is unfortunate because there is no myositis and no ossification in muscles The condition is quite different from the congenital abnormality "myositis ossificans progressiva" in which bone is actually laid down in the muscle bellies

TYPES OF PATHOLOGICAL OSSIFICATION

Myositis ossificans progressiva—This is a disorder of congenital origin in which there are recurrent inflammatory attacks in fibrous tissue planes with ossification in tendons and the fibrous intersections of muscles after each such attack[1,2] (Figs 115-116) All skeletal muscles are involved the spine becomes quite rigid and every affected joint is ankylosed There is almost constant association with congenital shortening of the great toe The disease progresses steadily and it is not amenable to any known treatment

Heterotopic ossification—A second type of pathological bone formation must also be distinguished from traumatic ossification It occurs in a localised form in tissues remote from the skeleton and from periosteum The same connective tissues in which pathological calcification is observed are the most common sites of heterotopic ossification It has been reported in the semilunar cartilages of the knee joint, in abdominal scars and in the tendo Achillis[3] (Fig 117) As with myositis ossificans progressiva, the bone formation does not arise as the direct result of injury, and it is beyond the control of the surgeon

"*Myositis ossificans traumatica*" (traumatic subperiosteal ossification)—The traumatic condition, on the other hand, is entirely within the control of the surgeon It is an avoidable complication, and its progress can always be arrested[4,5] The old theory that osteoblasts escaped from the bone and wandered into muscles, laying down bone in their course, is erroneous New bone formation occurs only within the limits of displaced periosteum and it is nothing more than the ossification of a subperiosteal hæmatoma[6] If displacement of the periosteum is prevented, and extensive hæmatomata are not allowed to occur, the complication is never seen

[1] Hutchinson, H "Multiple Exostoses with Ossification of Fascia" *Arch Surg*, 1896, 7, 138
[2] Frejka, B "Myositis ossificans progressiva" *J Bone Joint Surg*, 1929, 11, 157
[3] Watson-Jones, R, & Roberts, R E "Calcification, Decalcification & Ossification" *Brit J Surg*, 1934, 21, 461
[4] Watson-Jones, R "Myositis Ossificans of Quadriceps" *Brit med J*, 1930, 2, 592
[5] Watson-Jones, R *Canad med Ass J*, 1931, 24, 803
[6] Greig, D M "Traumatic Osteoma of Long Bones" *Surg Path Bone*, 1931, 170 Edinburgh Oliver & Boyd Ltd



FIG. 115



FIG 116

Myositis ossificans progressiva

Typical ossification in the fibrous intersections of the spinal muscles

(*By courtesy of Sir Thomas Fairbank—Mr Jackson Burrows' case*)



FIG 117

Heterotopic ossification of the tendo Achillis occurring twenty years after tenotomy of the tendon

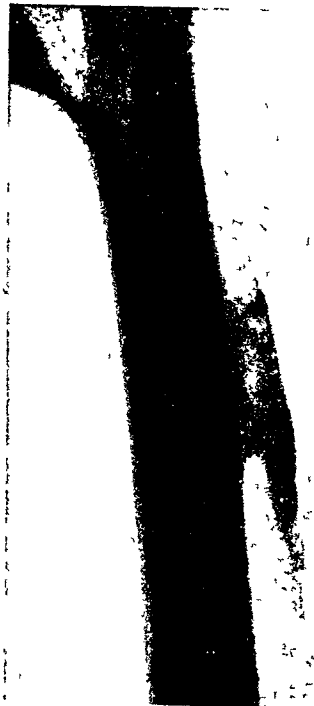


FIG 118

Traumatic ossification of subperiosteal hæmatoma of the femoral shaft

TRAUMATIC SUBPERIOSTEAL OSSIFICATION

Pathology—If periosteum is lifted from bone, new bone is always formed in the organising granulation tissue beneath the displaced periosteum. This is shown in Figure 119, a section taken thirty-five days after injury to the lower shaft of the femur in a boy aged four years The femoral cortex is the dark shadow in the lower field, it is separated from the horizontal fibres of the periosteum in the upper field by an area of organising granulation tissue in which woven bone is being laid down in the subperiosteal area. Elevation of the periosteum leads inevitably to subperiosteal ossification, and this is the essential pathology of traumatic ossification which has so unfortunately become known as "myositis ossificans"—a title that bears no relation whatever to the pathological or the clinical findings

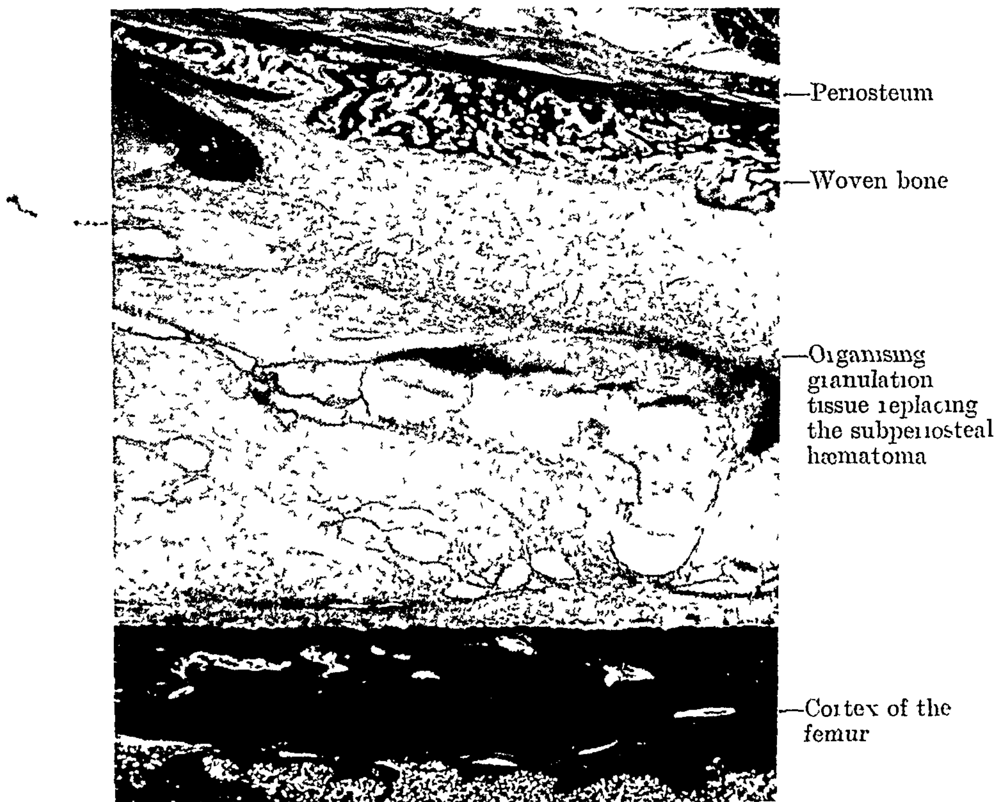


FIG 119
Traumatic subperiosteal ossification in a child aged four years (section prepared in the Bernard Baron Institute of the London Hospital, by courtesy of Prof D Russell).

Quadriceps avulsion with ossification—If a young or middle-aged patient stumbles so that his knee joint is flexed forcibly by body weight and at the same time he tries to save himself by strongly contracting the quadriceps muscle, the opposing strains cause a fracture of the patella When a more elderly patient suffers a similar strain, the insertion of the quadriceps muscle may give way instead of the bone The quadriceps is avulsed and it tears the limiting fibrous membrane from the upper border of the patella If the muscle is stitched back so that it is replaced in accurate contact with the bone, recovery may be complete (Fig 120). If the injury is treated without operative

suture, by simple immobilisation of the knee on a back splint the quadriceps remains retracted an inch or more from the rawed bone the interval is filled with blood clot bounded by displaced periosteum This subperiosteal hæmatoma undergoes gradual absorption, but at the same time new bone is formed within it, the two processes go on simultaneously. Provided that immobility is continued, the volume of the final mass of bone is considerably smaller than the original hæmatoma, and moreover it becomes firmly attached to the patella (Fig 121). Finally, if the injury is treated neither by operative suture nor by the immobilisation of a back splint, but the joint is regularly exercised and forcibly flexed, the detached periosteum is ruptured and the subperiosteal hæmatoma is disseminated Absorption is delayed and there may even be fresh hæmorrhage; the new bone is



Fig 120



Fig 121



Fig 122

Three cases of avulsion of the quadriceps from the patella

In the first case, shown in Figure 120, the muscle was at once sutured to the bone and was accurately replaced, so that there was no abnormal ossification In the second case, shown in Figure 121, no operation was performed, but the knee joint was immobilised on a back-splint, bone formed only in the hæmatoma within the hood of avulsed membrane and it united to the patella In the third case, shown in Figure 122, early movement and passive stretching was permitted, the hæmatoma was disseminated and scattered bone was formed, not fused to the patella, thus giving rise to the condition known as "myositis ossificans"

scattered irregularly above the patella (Fig 122) This is the type of bone formation that is described as "myositis ossificans of the quadriceps" Clearly, however, the ossification is still within the periosteum, and the question as to whether there will be no bone formation at all, a localised mass attached to the patella, or an extensive formation of scattered bone, depends entirely upon the treatment.

The same pathology explains traumatic bone formation on the surface of the shaft of the femur, when the quadriceps muscle is avulsed and bone is formed within the new limits of the periosteum (Fig 118) It explains the bone formation that may follow injury to the ankle, knee, shoulder or elbow joints, and indeed to any joint where periosteum is detached by the avulsion of muscles, tendons, ligaments or joint capsules

Traumatic ossification at the ankle joint—The anterior capsule of the ankle joint may be torn from the neck of the talus by forcible plantar flexion.

If the foot is immobilised in dorsiflexion the capsule is reattached with minimal new bone formation ; but if the foot is plantar-flexed the periosteum is held away from the talus and a bone spur develops which may limit dorsiflexion movement. If the ankle is exercised and passively stretched, a considerable mass of irregular new bone is formed in front of the joint This bone formation is often seen in football players from the repeated minor strains of kicking a heavy ball with the dorsum of the foot.

Traumatic ossification at the knee joint—When the medial collateral ligament of the knee joint is torn in the middle of its fibres, the hæmatoma does not communicate with any bone and repair takes place without ossification. If the ligament is avulsed at its proximal end from the medial femoral condyle, the hæmatoma is subperiosteal and must undergo ossification The degree of new bone formation, and the localisation or scattering of it, depends on the treatment It is only the failure to recognise and to understand the pathology of the condition that has led authors to describe this as a special clinical entity with the title of "Pellegrini-Stieda's disease"[1-3] It does not differ in any respect from the ossification of any traumatic subperiosteal hæmatoma



Fig 123

Subperiosteal ossification of avulsed conoid and trapezoid ligaments due to unreduced upward and backward dislocation of clavicle



Fig 124

Traumatic ossification round shoulder joint due to passive stretching after dislocation of the joint

Traumatic ossification at the shoulder—When the clavicle is dislocated at its outer end there is tearing of the acromio-clavicular ligaments, and avulsion of the conoid and trapezoid ligaments from the coracoid process If the dislocation is reduced and immobilised the ligaments become reattached , but if the dislocation is not reduced, the subperiosteal hæmatoma at the site of avulsion of each of these ligaments undergoes ossification (Fig 123) After traumatic dislocation of the shoulder joint early forcible passive movement may cause extensive subperiosteal ossification (Fig 124)

Traumatic ossification at the elbow—It is in the elbow joints of children that the complication is most often seen This is simply because periosteum

[1] Odessky, I N "The Koehler-Pellegrini-Stieda Syndrome" *Lyon Chir*, 1937, **34**, 272
[2] Callen, H S "Pellegrini-Stieda s Disease Manifestation in Knee of Post-traumatic Changes Common to other Joints" *Radiology*, 1937, 29, 158
[3] Brumbaugh, H L "Calcifying Tendinitis Traumatica" *Amer J. Surg*, 1940, **48**, 681

is more easily stripped in the child than in the adult, and because in children the elbow joint is dislocated more often than any other joint. Obviously the condition will be more frequent after dislocations than fractures, because muscle and capsule insertions into periosteum must necessarily be avulsed in a dislocation. The two causes of ossification of the subperiosteal hæmatoma after elbow dislocation are: (1) failure to reduce the dislocation promptly—the longer the bones are displaced the longer the periosteum is displaced, and the more advanced is the ossification of the hæmatoma, and (2) passive stretching and forcible vigorous movements soon after reduction of the dislocation and before the periosteum is firmly reattached to the bone.

It must be emphasised that it is *passive* stretching and not *active* exercise that redisplaces the periosteum. It is the deliberate stretching of a stiff joint by an enthusiastic masseuse or anxious parent, or by the drag of heavy buckets of water or bags of sand, that is responsible The joint has usually been immobilised in flexion, and therefore it is extension movement that is limited and may be forced The subperiosteal ossification is seen in front of the joint, sometimes from avulsion of the brachialis anticus from the ulna, sometimes from avulsion of the forearm muscles from the condyles,



FIG 125

Subperiosteal ossification due to passive stretching after supracondylar fracture Prolonged immobilisation would cause permanent stiffness because the joint has been seriously traumatised.



FIG 126

Same case as Figure 125, treated without immobilisation. Passive stretching was prohibited but active exercise encouraged. The bone has consolidated just as rapidly as if the joint had been immobilised.

and sometimes from avulsion of both When both have been avulsed, subsequent tearing of the periosteal walls of the hæmatomata may allow them to communicate, and the joint then becomes ankylosed by a continuous bridge of bone.

Treatment of traumatic ossification at the elbow—The first sign of this complication is the radiographic evidence of a cloudy shadow (Fig. 125) The shadow gradually becomes more dense, but since the hæmatoma is continually absorbing, the final bony mass is much smaller than the original shadow (Fig 126). The one essential treatment is to stop the passive joint stretching which was responsible There must be no massage or any other form of treatment by a masseuse The child must not be permitted to carry heavy weights, or hang from overhead beams by the affected limb. Movement must be allowed to recover at its own rate by the patient's own guarded activity

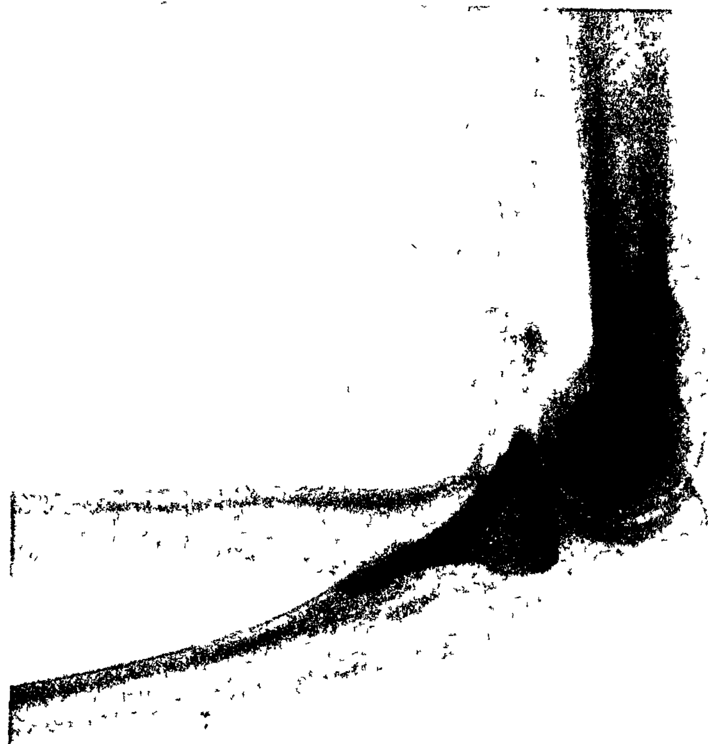


FIG. 127
August 1944

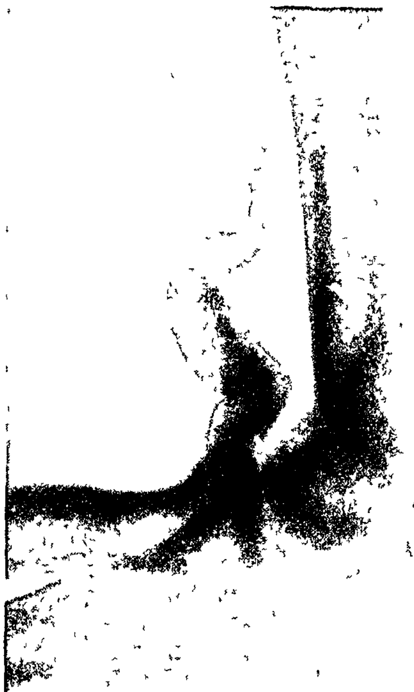


FIG 128
November 1944



FIG 129
January 1945

Traumatic ossification at the elbow due to avulsion of the biceps and brachialis anticus The subperiosteal hæmatoma has ossified and formed an osteoma attached to the coronoid process Note that there has been steady absorption of the ossifying hæmatoma so that the final mass is much smaller than the original shadow.

The absolute immobility sometimes recommended is neither necessary nor advisable Avulsion of the periosteum and hæmatoma formation is the result of passive stretching, not of active exercise The only treatment necessary is to prohibit passive stretching. Recovery is no more rapid if active exercise is also prohibited by completely immobilising the joint in plaster On the contrary this complete immobility allows consolidation of the adhesions which have also occurred from the passive stretching, and permanent stiffness and limitation of extension are then inevitable If the patient's own activity is permitted, the hæmatoma will still absorb the new bone will still shrink, and yet mobility will gradually improve.

Operative removal of bone spurs and bridges which are locking the joint must not be undertaken in the early stages. An operation performed before the ossifying hæmatoma is absorbed will only disseminate it and add to the ossifying area the new hæmatoma of operation. There is no indication for operation until the bone is finally consolidated, and until spontaneous recovery of movement is shown by actual measurement to have ceased. If a bone spur is obstructing movement it may then be removed, but very often the stiffness is due to the adhesions that were also caused by the stretching rather than to the bone block, and the range of movement may not be improved by operation.

Rare type of traumatic ossification at the elbow—A subperiosteal hæmatoma at the elbow joint may arise from disinsertion of the biceps tendon even when there has been no dislocation or fracture.[1] The periosteum of the radius is stripped and retracted several inches by the elastic recoil of the muscle If it is not reattached, an extensive subperiosteal hæmatoma remains and undergoes ossification. I have seen three such cases. In one, the injury was sustained by a man who "shook hands with a horse by flexing his elbow and placing his supinated forearm below the animal's raised forelimb At the moment that the biceps was tense the horse decided to shake no more, put his foot to the ground and disinserted the muscle The retracted tendon was exposed through a 1-inch incision in the arm, and through an incision over the back of the interosseous space long forceps were introduced by which the tendon could be pulled down and sutured to bone In this way the hæmatoma was obliterated without an extensive dissection in front of the joint. Recovery was complete and there was no trace of the bone formation usually seen after this injury One other case of traumatic ossification from avulsion of the biceps tendon which had not been recognised until ossification of the hæmatoma was well advanced, is shown in Figures 127-129

[1] Sénèque, J, and Berthe, R "Ruptures and Disinsertions of Distal Tendon of Biceps" (report of one case, abstracts of literature) *J Chir, Paris*, 1935, **46**, 347

CHAPTER V

AVASCULAR NECROSIS OF BONE

Surprisingly little attention has been paid in fracture treatment to the problems of avascular necrosis of bone and articular cartilage. Every group of fractures may be influenced by this complication—incapacity periods may be extended from months to years, and permanent disability may take the place of complete recovery It is of the most vital importance



Fig 130

Sequestration of upper femoral epiphysis in acute septic arthritis of the hip joint The epiphysis has not participated in neighbouring decalcification and it is therefore avascular

Pathology of avascular aseptic necrosis—It was König in 1888 who described the pathology of osteochondritis dissecans as "quiet necrosis" This was probably the first recognition of what is now known as aseptic or avascular necrosis The blood supply of the bone may be lost through embolism or thrombosis of its vessels[1 2] or it may be cut off by a fracture[3 5] The pathological changes that supervene may be described in three stages

Onset of necrosis—There is immediate cellular death of the avascular tissues The marrow elements change to a formless oily debris, bone cells disintegrate and lacunæ become empty tombs. When a joint surface is involved, patches of necrosis appear, separated by areas where articular cartilage has survived by direct nutrition from the synovial fluid. The general architecture of the bone remains undisturbed so that the radiographic appearances are unchanged Soon, however, neighbouring bone reacts with an active hyperæmia which is manifested in osteoporosis of the living bone. This osteoporosis can occur only in the presence of a free blood

[1] Phemister, D B "Repair of Bone in the Presence of Aseptic Necrosis from Fractures Transplantations and Vascular Obstruction." *J. Bone Joint Surg*, 1930 12, 769
[2] Coles B L, and Moore, M "Caisson Disease in Bones and Joints" *Ann Surg*, 1940, 111, 1065
[3] Watson-Jones R, & Roberts R. L "Calcification, Decalcification & Ossification" *Brit J. Surg*, 1934, 21, 461.
[4] Watson Jones R and Roberts R L "Pathological Calcification and Ossification" *Proc Roy Soc Med*, (Section of Radiology), 1932-33, 26, 851
[5] Watson Jones R and Roberts, R L *Brit J Radiol*, 1934, 7, 321

supply. Avascular bone cannot be deossified; it retains its original calcium content. It is for this reason that a sequestrum can be distinguished radiographically in infections of bone (Fig 130), and whether the blood supply is cut off by infection or by injury the same differentiation between dead and living bone becomes possible. The necrotic area preserves its original density and, by contrast, it may even appear to be increased in density.

Stage of regeneration—Active hyperæmia of the neighbouring bone initiates growth of granulation tissue Capillary loops and a fibrous stroma invade the necrotic area Phagocytes, multinuclear and mononuclear, resorb the dead marrow and grow along the Haversian canals which become enlarged to several times their original size They are followed by bone-forming cells, and bone resorption and bone formation go on almost simultaneously The Haversian canals are rebuilt, layer upon layer, and they gradually resume their normal proportions: both the form and the architecture are preserved The process is identical with that by which a bone graft, cut off from its circulation, is invaded and replaced by living bone. The replacement may be traced radiographically by the porosis that accompanies revascularisation In earlier stages the appearances often suggest fragmentation of dead bone, because tongue-like inroads of vascular granulation tissue surround the islets of avascular dead bone Similarly, localised areas of bone resorption may give an appearance of cyst formation.



Fig. 131

Microphotograph of the proximal half of a fractured carpal scaphoid bone, showing avascular necrosis (Van Giesen, ×75) The bone lacunæ are empty, proof of death of the cells. New woven bone is being laid down by which the dead bone will be replaced (the gap between the new woven bone, stained red, and the dead bone, stained yellow, is an artefact in cutting the section)

Stage of healing—Regeneration occupies many months or even years. The newly formed bone is soft and easily distorted, but ultimately it regains the full strength of original bone Articular cartilage, on the other hand, may suffer more permanent damage It is largely replaced by fibrous tissue and fibro-cartilage Early weight-bearing encourages collapse of the subchondral bone and it may be responsible for irregularity of the joint contours Even if there is freedom from weight-bearing the new fibro-cartilage is so imperfect that degenerative arthritis often develops

Clinical applications of avascular necrosis—In every fracture minute detached fragments of bone are deprived of their blood supply Indeed



Fig. 132



Fig 133



Fig 134

Fracture neck of femur At the time of injury both fragments are equally calcified (Fig 132) Seven weeks later there is disuse porosis of all bones except the femoral head, which is therefore avascular (Fig 133) Ten weeks later the evidence has disappeared for there is no longer porosis of the other bones (Fig 134) An accurate prognosis is possible only if radiographs are taken during the second month

over the whole fractured surface bone cells die, lacunæ are empty, and there is a thin film of avascular necrotic bone This is invaded and replaced by living bone and it represents the normal process of repair (see Figs 1-4). In the treatment of non-union of a fracture, the graft is avascular necrotic bone which is gradually replaced by living bone. Similarly, when a recent fracture is reduced by open operation, considerable areas of bone may be stripped of periosteal and vascular attachments. Even the whole thickness of the shaft of a long bone may be rendered avascular, become necrotic, and be replaced by new bone In short, complete loss of the blood supply to one fragment is no barrier to union. Repair is delayed and it is sometimes complete only after twelve months, two years, or even longer Nevertheless, if immobility is continued long enough, sound union of the fracture will be achieved.

If the avascular fragment carries with it the articular cartilage of a joint the outlook is wholly changed. Repair will be slow, but more—it will be imperfect. Replacement of hyaline cartilage with fibrous tissue, or with an imperfect fibro-cartilage, is almost inevitable. Very often the joint space becomes narrowed movement is restricted and degenerative arthritis supervenes.

Early radiographic diagnosis of avascular necrosis—Early radiographic diagnosis is based upon the apparent density of avascular bone [1] Since the bone has no blood supply, its calcium content cannot change appreciably; whereas neighbouring vascular bone is resorbed and becomes porotic. In these circumstances, avascular bone appears dense by contrast It must be recognised, however, that the density is purely relative, that it is not a true hypercalcification, and that the evidence is not available unless neighbouring bone shows porosis. The diagnosis cannot be made during the first few weeks after injury before disuse porosis has begun. Moreover, it cannot be made in the later stages after the resumption of functional activity The whole tendency of modern fracture treatment is to minimise disuse change by early functional activity, and the sign of relative density is transient (Figs. 132-134). It is true that after several months other radiographic signs of avascular necrosis may appear, particularly in the lower limb where the fragility of necrotic bone is shown by disintegration and crushing under the pressure of weight-bearing. But the diagnosis should be made before this stage is reached so that the harmful effects of early weight-bearing can be avoided Moreover, bone disintegration and degenerative arthritis do not always develop so quickly; there is sometimes an interval of several years before the onset of these sequelæ, and if the passing shadow of relative density has been overlooked a completely erroneous prognosis may have been given. It is important, therefore, when fractures are sustained in regions where avascular necrosis may supervene, to take radiographs during the second, third and fourth months, at not more than monthly intervals If the shadow of relative density appears in any one of these films, the warning must not be ignored

Unusual fracture of the patella—The pathology, radiographic diagnosis and clinical significance of avascular necrosis of bone were demonstrated clearly in an unusual fracture of the patella sustained by an aircraft mechanic who fell from an engine stand Seven days after injury the bone was exposed

[1] Watson-Jones, R, and Roberts, R E "Significance of Density of Bone Shadow" *Brit J Surg*, 1934, 21, 467.

at operation and the fragments were fixed in accurate apposition by means of a vitallium screw driven through the lower pole. The wound healed by first intention, but six weeks later radiographs showed striking evidence of relative density of the proximal fragment which had failed to share the post-traumatic osteoporosis that was so evident in the distal fragment (Fig. 135). It was obvious that the proximal fragment had lost its blood supply, possibly in consequence of wide stripping of soft tissues in the course of operative reduction, and since degeneration of the overlying articular cartilage was inevitable, and would probably have caused patello-femoral arthritis, the patella was excised and the quadriceps tendon repaired Sagittal section of the whole bone shows resorption of trabeculæ in the living distal fragment, but persistence of normal thickness and density of the trabeculæ of the proximal fragment (Fig 136) These trabeculæ consist of dead bone, and one is shown in high magnification in Figure 137 The cells in the bone spaces have undergone lysis after death, so that the bone spaces are empty (note the "cement lines" between three systems in the dead trabecula) Below are osteoclasts, actively engaged in resorption of the dead bone Above the trabecula is a zone of newly formed bone with living bone cells, and adjacent to it a continuous layer of deeply stained osteoblasts which are not resolved in this section into individual cells except on the extreme left, where a few are cut obliquely and appear as parallel oblongs. The marrow above and below the trabecula, though infiltrated with lymphocytes, is alive. This field is typical of the whole of the proximal fragment of the patella The marrow has survived, but every single bone trabecula is dead and is undergoing osteoclastic resorption and osteoblastic replacement This process of regeneration has been described as "creeping substitution", but "creeping" is hardly the word. It is occurring almost uniformly throughout the whole fragment and at a fast rate Moreover, bone is being laid down on the surface of every trabecula and therefore in the pattern of the original bone It is only a matter of time before perfect reconstitution of the bone will be complete On the other hand, the cells of the articular cartilage of the proximal fragment have also died; they are swollen and show reversal of normal staining reactions so that the nuclei are acidophil and the cytoplasm basophil (Fig 138) Imperfect regeneration of the dead articular cartilage is almost inevitable

AVASCULAR NECROSIS OF THE HEAD OF THE FEMUR

Blood supply of the femoral head—The nutrient and periosteal vessels of the femoral shaft extend as high as the trochanteric region and lower part of the neck but they do not contribute to the blood supply of the head of the femur The femoral head is supplied from two sources (1) capsular or retinacular vessels, (2) fovealar vessels of the ligamentum teres The vessels of the capsule run on the surface of the neck in retinacula reflected from the deep aspect of the capsule, finally entering the bone in the subcapital region and then supplying the upper part of the neck of the femur and the greater part of the head The fovealar vessels of the ligamentum teres are well defined in children and usually remain patent or even increase in



Fig 135

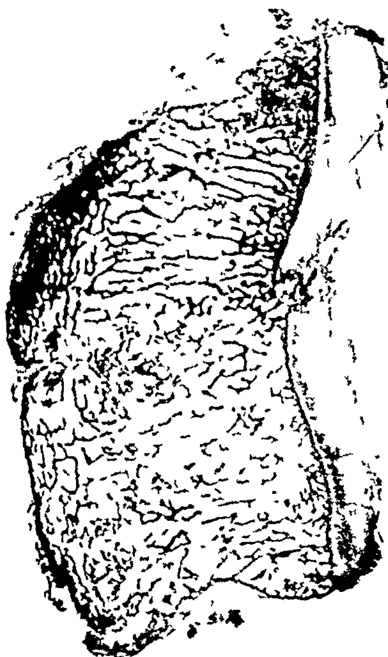


Fig. 136

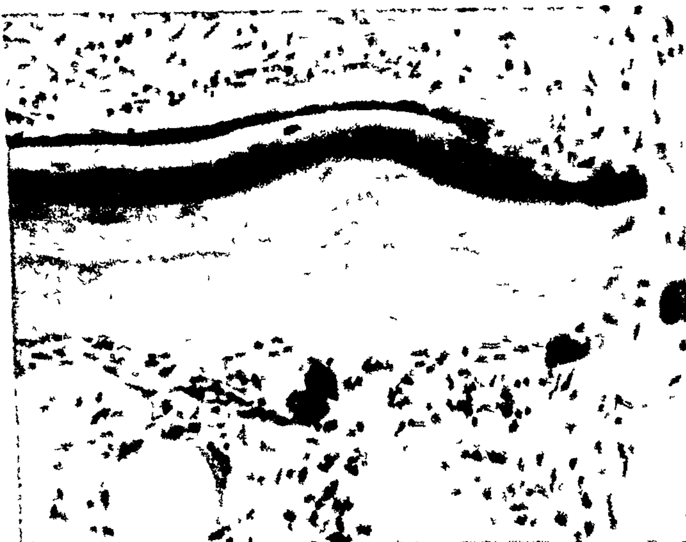


Fig 137

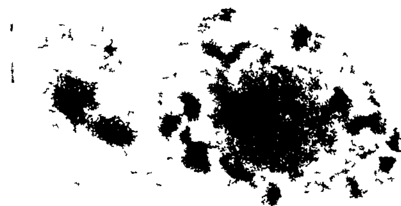


Fig 138



Fig 139

Fracture of the patella six weeks after operative reduction and internal fixation by vitallium screw, showing relative density of the proximal fragment (Fig 135) The patella was excised and the specimen is shown in Figure 136 One trabecula from the dead proximal fragment, indicated by the rectangle marked in white, is seen magnified ×195 in Figure 137 The dead trabecula with empty bone spaces is undergoing resorption by osteoclasts below It is being replaced by new bone laid on its surface above—the zone of deeply stained osteoblasts is not resolved in this section into individual cells but appears as a continuous dark line immediately above the new bone The marrow above and below, though infiltrated with lymphocytes, is alive Figure 138 shows one cell of the articular cartilage of the dead fragment (×1450), note the swelling and reversal of normal staining reactions (the nucleus being acidophil and the cytoplasm basophil), compare with two living cartilage cells from the distal fragment (Fig 139)

size in adult life[1-4]; they supply an area of bone and articular cartilage in the region of the fovea centralis There is seldom a true anastomosis between these two sets of arteries, and if either group of vessels is obliterated by injury, thrombosis or embolism the corresponding part of the head of the femur may lose its blood supply[5] The capsular vessels may be interrupted in the capsule itself as the result of traumatic dislocation, or by surgical exposure of the joint, or they may be damaged in their retinacular course on the surface of the femoral neck by fractures in the upper part of the neck The vessels of the ligamentum teres may be torn by the injury of traumatic dislocation of the hip, or they may

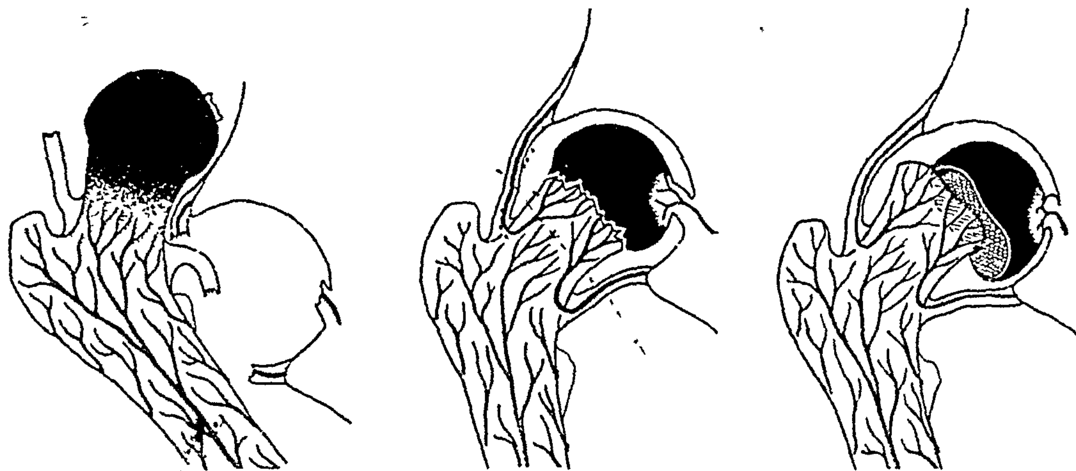


FIG 140

The femoral head is supplied with blood by vessels from the capsule If these are damaged by a traumatic dislocation, a high fracture of the neck, or epiphyseal separation, avascular necrosis supervenes

be damaged by forcible rotation movements or manipulation of the joint causing stretching of the ligament over the margin of the acetabular fossa.

Fracture of the neck of the femur[6]—*Basal fractures* in the trochanteric region of the femur lie below the site of entry of capsular vessels and do not interfere with the blood supply of either fragment *Transcervical fractures* occur in a dangerous region. Capsular retinacula usually remain attached to the proximal fragment but the blood supply they carry is precarious. If, for example, the fracture is not immobilised promptly by nailing the fragments, unguarded movement may damage the remaining vessels. Forcible manipulation during reduction of the fracture may be no less dangerous Fractures at this level are more liable to interfere with the blood supply of the head in children than in adults Even low fractures near the basal region often cause avascular necrosis in children.[7] *Subcapital fractures*—A high subcapital fracture may lie above every capsular

[1] Chandler, S. B., and Kreuscher, P. H. "Blood Supply of Ligamentum Teres." *J. Bone Joint Surg.*, 1932, 14, 834
[2] Wolcott, W. E. "Circulation of Head and Neck of Femur." *J. Amer. med. Ass.*, 1933, 100, 27.
[3] Kleinberg, S., and Friedman, L. "Vascularity of Ligamentum Teres." *Bull. Hosp. Joint Dis.*, 1940, 1, 72
[4] Tucker, F. R. "Arterial Supply of the Femoral Head." *J. Bone Joint Surg.*, 1949, 31-B, 82.
[5] Watson-Jones, R. "Avascular Necrosis in Fractures, Dislocations, Perthes' Disease, Coxa Vara, Congenital Dislocation and Osteoarthritis of the Hip." *Brit. med. J.*, 1942, 1, 503
[6] Santos, J. V. "Changes in the Head of the Femur after Intracapsular Fracture." *Arch. Surg.*, 1930, 21, 470
[7] Seddon, H. J. "Necrosis Head of Femur following Fracture in Child." *Proc. Roy. Soc. Med.*, 1936, 30, 210.

attachment to the head, no matter what precautions are then taken, the blood supply is lost to all parts of the femoral head (Fig 142), except sometimes in the region of the fovea (Fig 141) *Operative exposure of femoral neck fractures*—Open exposure of any fracture of the neck of the femur, with division and retraction of the capsule, endangers the blood supply of the head Vessels which escaped damage by the bone injury, and on which the femoral head depends for its survival, may sustain injury in the capsular part of their course For this reason, operative exposure of the fragments has now been abandoned, and nailing of the fracture is performed by an "extra-articular technique" through an incision over the trochanter with radiographic control



Fig. 141
The part of the femoral head supplied by capsular vessels is relatively dense and therefore avascular The part supplied by the ligamentum teres is deossified and therefore vascular

Dislocation of the hip joint—Traumatic dislocation of the hip joint is necessarily accompanied by rupture of the ligamentum teres and loss of blood supply to part of the head of the femur in the region of the fovea. Furthermore, the capsule of the joint is also torn, and according to the degree of violence this source of blood supply is either impaired or completely lost Avascular necrosis with degenerative arthritis occurs in a high proportion of traumatic hip dislocations The incidence is increased if further injury to surviving capsular vessels is inflicted by unduly forcible manipulation, early passive movement, or operative reduction with stripping and division of the capsule



Fig 142
The whole femoral head is dense. Capsular vessels have been interrupted by the fracture, and fovealar vessels of the ligamentum teres are inadequate Inset—A Lorenz osteotomy is better than nailing if the femoral head is avascular.

Epiphyseal coxa vara—Separation and backward displacement of the upper femoral epiphysis may be due to a single acute injury, but more often it develops gradually in heavy children who suffer from pituitary dystrophies As the epiphysis rotates backwards on the neck, its central position in the acetabulum is maintained by increasing external rotation deformity of the limb In former days it was customary to attempt reduction by forcible correction under anæsthesia If the limb is strongly rotated inwards, and correction

does not take place between the femoral neck and epiphysis, the epiphysis is twisted into the back of the joint, the ligamentum teres is stretched over the margin of the acetabular fossa, and the vessels within the ligament are injured. If the position is maintained for several weeks or months by a plaster spica, destruction of the fovealar vessels is almost inevitable This mistake was often made in the past because, if antero-posterior radiographs are taken with the limb in strong internal rotation, a radiographic illusion gives an appearance of accurate reduction even when the epiphysis is still displaced widely (see *Volume II*). Avascular necrosis supervened with degeneration of the articular cartilage, narrowing of the joint space, stiffness of the hip and arthritis. This complication can usually be avoided if displacement of the upper femoral epiphysis is corrected only by cautious and gradual traction without anæsthesia



Fig 143



Fig. 144

Low transcervical fracture in a child, causing avascular necrosis This complication occurs with very great frequency in fractures of the femoral neck of children—much more frequently than in the corresponding fractures of adults

Legg-Perthes' disease of the hip (pseudo-coxalgia)—When fracture of the neck of femur or dislocation of the hip joint occurs in young children the complication of avascular necrosis is manifested by relative density of the epiphysis, followed after several months, when revascularisation is taking place, by an appearance of fragmentation and, if weight-bearing is permitted, by flattening of the contour of the femoral head (Figs 143-144) These epiphyseal changes, attributable to rupture of the ligamentum teres or interruption of the capsular blood vessels, are strikingly similar to the changes of Legg-Perthes' disease of the hip joint So strong is the clinical resemblance that it must be assumed that Legg-Perthes' disease is a simple avascular necrosis due to concealed injury or thrombosis of the vessels of the ligamentum teres or capsule

Congenital dislocation of the hip joint—In former days, congenital dislocation of the hip joint was reduced by forcible methods The violence of the manipulations damaged the vessels of the ligamentum teres, thus explaining the frequency with which typical Legg-Perthes' disease supervened Operative exposure of congenital dislocations was complicated in a similar way, and the operation of reconstructing the upper lip of the acetabulum by the insertion of a bone graft fell into disrepute for this reason In these cases it was the retinacular vessels of the capsule rather than the fovealar vessels that were damaged, but the sequel was the same Orthopædic surgeons now reduce congenital dislocations with great gentleness and the complication of Legg-Perthes' disease is seldom seen Nevertheless there may still be danger if the limb is immobilised in plaster in the extreme position of 90° abduction and 90° flexion It has been shown by Tucker[1]

[1] Tucker, F R "Arterial Supply of the Femoral Head" *J Bone Joint Surg*, 1949, **30-B**, 82

that in this position the most important group of capsular vessels—the postero-superior retinacular vessels which are present in 100 per cent. of children and adults, and are two or three times the size of any other capsular vessels—may be compressed between the femoral neck and the acetabular margin

Monarticular osteoarthritis of the hip joint—In the same way that a vulnerable blood supply explains the frequency of Perthes' disease of the hip joint in children, it explains the frequency of osteoarthritis of the hip joint in adults. Osteoarthritis occurs far more commonly in this joint than in any other The radiographic appearances are similar to those of degenerative arthritis from avascular necrosis after dislocation of the joint, there is the same mottling irregular density, cyst formation, narrowing of the joint space, and flattening of the contour of the head When exposed at operation, areas of avascular necrotic bone and articular cartilage are evident Is it not probable that the usual cause of osteoarthritis of the hip joint is avascular necrosis? The source of deprivation of the blood supply may be obvious, as when there is a fracture or dislocation, or concealed, as from minor strains and twists which injure the ligamentum teres and cause thrombosis of its vessels

Summary of Avascular Necrosis of the Head of the Femur

1 Retinacular blood vessels of the capsule of the hip joint, and fovealar vessels of the ligamentum teres, are each important in maintaining the nutrition of the femoral head

2 If either of these groups of blood vessels is destroyed, avascular necrosis of the femoral head may supervene and cause degenerative arthritis in the adult or epiphyseal disturbance in the child

3 Capsular vessels may be destroyed by injury to the capsule from traumatic dislocation, forcible manipulation, immobilisation of congenital dislocation in extreme abduction, or the surgical exposure of fractures.

4 Capsular vessels may be destroyed by subcapital fractures in adults or transcervical fractures in children

5 Fovealar vessels may be destroyed by rupture of the ligamentum teres in traumatic dislocation of the joint, or by stretching the ligament by forcible internal rotation in epiphyseal coxa vara or congenital dislocation.

6 Legg-Perthes' disease in children and osteoarthritis of the hip joint in adults are manifestations of avascular necrosis. The deprivation of blood supply may be due to the obvious causes of fracture or dislocation, or to the concealed causes of minor injury or inflammatory thrombosis

FRACTURE OF THE NECK OF THE FEMUR

Sir Astley Cooper wrote of fracture of the neck of the femur in 1823: "It is impossible even for a few hours to preserve exact apposition of the fragments, and the surgeon should not be held responsible for a fracture over which he has so little control" Control by the surgeon was achieved with the introduction of the three-flanged steel nail by which exact apposition and complete immobility of the fragments could be maintained even when early

weight-bearing was permitted, a perfect result depended only upon the surgeon's skill in inserting the nail accurately. The recognition that impairment of blood supply might delay union for twelve months or two years, and that complete immobility of the fragments was needed throughout this time, made it still more obvious that nail fixation was the treatment of choice. But we now know that if the blood supply of the femoral head is so completely lost that avascular necrosis supervenes, a steel nail ploughs through the fragile bone, redisplacement occurs, the bone disintegrates and degenerative arthritis supervenes In these cases the nailing operation is no longer a brilliant success, it is better to adopt the second-best plan and perform an osteotomy of the Lorenz type in which an arthroplasty is established between femur and pelvis. the necrotic head of the femur remaining sequestered and immobile in the acetabulum (Fig 142 inset) What is the evidence on which the diagnosis of avascular necrosis may be established? When should the surgeon change his plan of treatment from the nailing operation to the osteotomy?

1. Relative density of the femoral head—If the fracture is several weeks or months old, so that limited function has given rise to the bone resorption of disuse, and the femoral head is relatively dense, impairment of its blood supply is proved In these circumstances the nailing operation should not be employed. On the other hand, in fractures a few days old the diagnosis cannot be made An accurate diagnosis could of course be established by deferring operation for several weeks and noting whether relative density of the head appeared, but as delay in immobilising the fragments may itself precipitate the complication by causing further damage to surviving vessels this course is not justifiable. The nailing operation should be performed without delay, radiographs being taken at monthly intervals If relative density is shown, even only in one film, the surgeon must be warned It does not necessarily follow that complete necrosis and rapid disintegration are inevitable (Figs 145-148). It is true that degenerative arthritis may supervene in the course of time, but meanwhile many years of good function may be enjoyed Two precautions should be taken. (1) Union will be slow, it will be complete only in twelve months, two years or even longer Throughout the greater part of this time stability of the bone will depend largely upon the nail, and it should not be removed even twelve or eighteen months after operation because immediate refracture will almost certainly occur (2) Weight-bearing should be deferred for many months in order to prevent crushing of the fragile bone and damage to the devitalised articular cartilage[1] Immobilisation of the joint is not advisable, revascularisation is promoted best by active non-weight-bearing exercise

2. Increasing penetration of the nail and shortening of the neck—Comparison of films taken at monthly intervals may show progressive shortening of the neck and increasing penetration of the nail (Figs. 149-151). The most reliable measurement is from the point of the nail to the articular surface Increasing penetration indicates abnormal fragility of bone and is evidence of necrosis. It is most pronounced when early weight-bearing

[1] Compere, E L, and Lee, J "Restoration of Physiological and Anatomical Function in Fractures of the Neck of the Femur" *J Bone Joint Surg*, 1940, 22, 261


Fig 145 Two months


Fig 146 Four months


Fig. 147 Eighteen months


Fig 148 Four years

Avascular necrosis with relative density of head, slow union of fracture and increasing penetration of nail

Nailed fracture of the neck of the femur two months after operation shows relative density of the head (Fig 145) Collapse and disintegration of the avascular necrotic head was prevented by delaying weight-bearing for eight months. Union was not complete until four years after operation (Fig 148) There was no increase in the penetration of the nail before weight-bearing was permitted (Figs 145-146), but some increasing penetration took place between the second and fourth years after operation (Figs 147-148)



FIG 149 Three months



FIG 150 Nine months



FIG. 151. Two years

Avascular necrosis proved by increasing penetration of the nail

Radiographs three months after operation suggested the possibility of relative density of the femoral head, but they were not conclusive (Fig. 149) Weight-bearing was permitted Six months later there is clear evidence of increasing penetration of the nail, proving avascular necrosis of the head (Fig 150) Two years after operation the nail has penetrated the joint surface and the femoral head has disintegrated (Fig 151)

has been permitted Ultimately the point of the nail may actually penetrate the joint and it is then necessary to remove it and, if the fracture has not already united to perform a Lorenz osteotomy.

3. Ploughing of the nail through the head—The nail may not only penetrate more deeply but may also plough upwards and forwards through the bone Comparison of antero-posterior and lateral radiographs at monthly intervals shows that the central position of the nail is not maintained (Figs 152-155) This must not be attributed to a fault in the nailing technique, reinsertion of the nail or the introduction of a longer nail is a mistake Redisplacement is occurring because the bone is too fragile to withstand the pressure of a nail The correct treatment is to remove the nail and perform a Lorenz osteotomy.

4. Slow union of the fracture—Slow union of the fracture, despite the complete immobilisation of a well-placed nail, can be accepted as evidence of impairment of blood supply If union is not complete within about six months, the head is avascular If, for example, a nail has been removed after six or twelve months and refracture has immediately occurred, a second nailing operation, or a nailing and grafting operation, is inadvisable. The head is avascular and necrotic, and an osteotomy should be performed.

5. Late onset of arthritis—If the sign of relative density was not conclusive and there has been no evidence of increasing penetration, ploughing of the nail, or slow union, the final evidence of avascular necrosis is often delayed by many years A slowly developing degenerative arthritis may supervene five, ten or even fifteen years after fracture (Figs 156-157). Removal of the nail, or replacement of the nail by a bone graft, appears to have no influence on this late development. Even the early insertion of a bone graft shortly after the fracture was sustained is of doubtful value in re-establishing the circulation [1] There is little or no support for the suggestion that the insertion of a bone graft serves as a channel for the development of new blood vessels.

Treatment of old un-united fractures—The question as to whether a fracture several months old should be treated by nailing and grafting, or by a reconstruction operation, depends entirely on the question of avascular necrosis If there has never been evidence of impairment of blood supply a six or twelve months' old fracture may be nailed successfully If, on the other hand, there is proof of necrosis of the head, even a six weeks' old fracture should not be nailed or grafted The fragile bone will probably not hold the nail and arthritis of the joint will supervene.

The type of reconstructive operation to be performed for old un-united fractures is also to be determined by the problem of avascular necrosis Many operations formerly described, such as the Brackett operation [2] in which the upper femoral shaft was implanted into the head, are now obsolete for this reason. The Whitman reconstruction operation,[3] in which the femoral head was removed and the stump of the neck implanted in the acetabulum, is likely to fail because stripping the neck deprives it of blood and induces avascular necrosis [4] The simple bifurcation osteotomy of Lorenz has superseded these procedures

[1] Phemister, D B "Pathology of Un-united Fractures of the Neck of the Femur with Special Reference to the Head" *J Bone Joint Surg*, 1939, **21**, 681
[2] Brackett, E G *Boston med Surg J*, 1917, **177**, 351, *J Bone Joint Surg*, 1938, **20**, 93
[3] Whitman, R *Surg Gynec Obstet*, 1921, **32**, 479, *J Bone Joint Surg*, 1933, **15**, 215
[4] Phemister, D B "Aseptic Necrosis of Bone" *J Bone Joint Surg*, 1930, **12**, 769



Fig. 152 One week



Fig. 153. Two months



Fig 154 Four months



Fig 155 Six months

Avascular necrosis proved by rapid ploughing of nail through the head.

Antero-posterior and lateral radiographs one week after operation showed that the nail was centrally placed (Fig 152) Despite protection from weight-bearing the nail ploughed through *the front of the head (Fig 153)* The head displaced backwards *into* the position *of coxa anteverta* (Figs 154-155). Even when the sign of relative density is inconclusive, redisplacement despite an accurately placed nail proves that the head is avascular, necrotic and fragile.



FIG. 156. One year.



FIG. 157. Eight years.

Avascular necrosis proved by late development of degenerative arthritis

This patient resumed heavy work within twelve months of operation (Fig 156) and for several years the result appeared to be perfect. Eight years after nailing there is clear evidence of disintegration of the head and degenerative arthritis, proving a slowly developing avascular necrosis (Fig 157).

TRAUMATIC DISLOCATION OF THE HIP JOINT

The frequency with which traumatic dislocation of the hip joint is complicated by avascular necrosis has not hitherto been recognised. The sequel of degenerative arthritis is sometimes delayed for several years,[1] and the dislocation and arthritis have not always been associated as cause and effect. In fact, however, a high proportion of traumatic hip dislocations are complicated in this way to a lesser or greater degree.[2] Rupture of the ligamentum teres causes localised necrosis of bone and cartilage in the region of the fovea, and in about 30 per cent. of cases additional injury to capsular vessels causes disintegration of bone and severe degenerative arthritis.

Diagnosis of avascular necrosis in children.—The complication may be recognised in early weeks by relative density of the upper femoral epiphysis, which does not undergo the bone resorption of disuse (Figs. 158-162). Subsequent changes are those characteristically seen in Legg-Perthes' disease. As revascularisation takes place, and islands of porotic bone appear, the epiphysis appears to be fragmented. If weight-bearing is permitted the fragile bone is crushed, the femoral head is flattened and the epiphysis and neck are broadened (coxa plana). Ultimately revascularisation is complete, the bone shows uniform ossification, but joint movement may remain limited by deformity of the head. Finally, ten or twenty years later, degenerative arthritis supervenes.

Treatment in children.—The essential treatment is to protect the necrotic bone from compression until revascularisation and regeneration are complete. Early weight-bearing is the worst treatment because it crushes the subchondral bone, distorts the joint surface, flattens the femoral head and interferes with regeneration of the articular cartilage. A plaster spica is of no particular value, and if the patient is allowed to walk in the plaster it is of no value at all. Even a walking calliper splint is ineffective. The patient must be recumbent and there must be traction on the limb. The traction should be continued until revascularisation is complete, usually for eighteen months or two years. For the first few months, while the joint is irritable and there is muscle spasm with a tendency to flexion-adduction deformity, the hip should also be immobilised by means of an abduction frame. For the next twelve or eighteen months traction should be continued either by suspended weights, or by tying the limb to the raised foot of the bed. Active hip movements should be encouraged. When the apparently dense bone has passed through the "fragmented" stage to the stage of completely uniform ossification, traction may be discontinued. A few months are then spent in more energetic non-weight-bearing exercise for increasing periods each day. Finally, after about two years, unprotected weight-bearing may be permitted.

Diagnosis of avascular necrosis in adults.—Every effort should be made to establish the diagnosis within two or three months of injury by the radiographic sign of relative density of the femoral head. If this evidence is inconclusive, the complication should be suspected on clinical grounds if it is found that rotation movement remains limited, and that flexion movement does not increase beyond 90°. In succeeding months the gradual

[1] Potts, J. N., and Obletz, B. L.: "Aseptic Necrosis of Head of Femur following Traumatic Dislocation." *J. Bone Joint Surg.*, 1939, 21, 101.
[2] Banks, S. W.: "Aseptic Necrosis of the Femoral Head following Traumatic Dislocation of the Hip." *J. Bone Joint Surg.*, 1941, 23, 753.



Fig. 158



Fig. 159 Three days



Fig. 160. Four months.



Fig 161. Ten months.



Fig. 162. Five years

Dislocation of hip joint in child with avascular necrosis—(pseudo-coxalgia).

Traumatic dislocation with fracture of pelvis (Fig 158) reduced by manipulation (Fig 159) Radiographs four months later show relative density of the area supplied by the vessels of the ligamentum teres (Fig 160) Typical changes of Legg-Perthes' disease (pseudo-coxalgia) supervened (Fig 161) but coxa plana was minimised by prolonged recumbency and traction (Fig 162)



FIG 163

Vitallium cup arthroplasty—Smith-Petersen technique Note that the new acetabulum is sunk deeply, and that there is free mobility of the cup within it There is also free mobility of the femoral head within the cup



FIG 164

Arthrodesis of hip for avascular necrosis and degenerative arthritis after dislocation of the joint (Fig 164), a three-flanged nail must be used to ensure consolidation in the optimal position (Watson-Jones technique, *J Amer med Assoc*, 1938, **110**, 278) It must be emphasised, however, that nail fixation alone, without operative denuding of articular cartilage and protection in a plaster spica for not less than four months. does not suffice Even after open arthrodesis, if the spica is discarded too soon, before consolidation is complete, pressure absorption causes loosening of the nail and failure of the arthrodesis (see Chapter XI)

development of flexion deformity confirms the diagnosis, and as years pass by the range of movements becomes steadily less The radiographic evidence may not be striking Comparison with the normal hip shows reduction in the joint space due to erosion of articular cartilage, and slight mottling of the head of the femur with irregular density and imperfect bone texture In other cases the disintegration of bone is much more rapid, the clinical diagnosis is obvious within twelve months or two years of injury, and radiographs show gross distortion, crushing and irregular density of the femoral head with complete disappearance of the joint space.

Treatment in adults—The treatment advocated for children, namely, recumbency and traction for two years, is seldom justified for adults. Not only is the adult unwilling to spend two years in bed but the regenerative powers of articular cartilage are less than in the child, and it is unlikely that the sequel of early degenerative arthritis can be avoided. It is better to insist on recumbency for the first three or four months after injury, and if severe arthritis with considerable pain and almost complete stiffness still develops, to arthrodese the joint. The alternative of arthroplasty may be considered, using the vitallium cup technique of Smith-Petersen[1] (Fig 163) But it should be recognised that a hip joint soundly ankylosed in the optimal position causes surprisingly little disability there is no appreciable limp : it is easy to walk ten or fifteen miles without discomfort or fatigue . patients can jump, hop, climb and run, play tennis and golf, enjoy mountaineering and even ski-ing : men go back to strenuous labour ; women do their normal household duties , there is no pain in the back there is little or no difficulty in sitting. The result is so completely reliable that to compete with it an arthroplasty must be quite perfect. It is, however, essential that ankylosis shall be sound and that it shall take place in the ideal position of strictly neutral rotation, with neither abduction, adduction nor flexion deformity. A long three-flanged nail must therefore be used, transfixing the upper femur and pelvis[2] (Fig. 164)

LEGG-PERTHES' DISEASE OF THE HIP

Traumatic dislocation of the hip joint and transcervical fracture of the neck of the femur in children may produce changes that are clinically and radiographically indistinguishable from Perthes' disease They are indistinguishable because they are the same This disease is simply an avascular necrosis of the upper femoral epiphysis. The frequency is explained by the vulnerability of the blood supply, which may be cut off by the injuries we have discussed, or by the capsular trauma of manipulative or operative reduction of congenital hip dislocations It may also be cut off by any strain or twist that causes thrombosis of capsular or fovealar vessels, or by the toxic and inflammatory changes that occur so often in the hip joints of children Whether the source of vascular deprivation is obvious as in fractures or dislocations of the joint, or concealed as in traumatic and toxic thrombosis, the sequelæ are the same

Many typical cases of Perthes' disease are already in the fragmented stage when first seen Continued weight-bearing has already crushed the

[1] Smith-Petersen, M N "Arthroplasty of the Hip" *J Bone Joint Surg*, 1931, 21, 269
[2] Watson-Jones, R "Arthrodesis of the Osteo-arthritic Hip" *J. Amer med Ass*, 1938, 110, 278



Fig 165



Fig. 166



Fig 167



Fig 168



Fig 169



Fig 170

Perthes' disease of hip on the first day of limping—no radiographic abnormality (Fig 165) Six weeks later, failure of decalcification of epiphysis proves avascularity (Fig 166) Figures 167-170 at four-monthly intervals show gradual revascularisation of epiphysis Traction has been continued throughout and there is therefore no crushing or flattening.

epiphysis so that it is flat and broad. This accounts for the characteristic coxa plana and wide femoral neck The appearances of density of the epiphysis and of fragmentation have been misunderstood We have believed that this was due to hypercalcification. Actually it is due to retention of the original calcium content while neighbouring bones have decalcified If a patient who ultimately develops the typical signs of Perthes' disease is seen on the first day of limping, the radiographic appearances are normal.[1] If the hip is immobilised so that diffuse porosis arises, and many radiographs are taken at weekly or fortnightly intervals, one film will show failure of decalcification of the epiphysis (Fig 166) This stage is transient and may easily be overlooked.

The correct treatment of Perthes' disease is the treatment of avascular necrosis of the femoral head in children. There must be traction for some months, and protection from weight-bearing for at least two years, until revascularisation is complete. Immobilisation in a plaster spica is worthless and a walking calliper splint is of doubtful value. Since in many cases, where there has been no actual dislocation or fracture, the loss of blood supply has not been complete, perfect recovery with a hip joint apparently indistinguishable from normal is often secured Nevertheless, as with any case of avascular necrosis of a joint surface, the regenerated articular cartilage is imperfect and degenerative arthritis may arise ten or twenty years later.

AVASCULAR NECROSIS OF THE HEAD OF THE HUMERUS

The shoulder joint is dislocated much more often than the hip joint but the injury is the result of relatively trivial violence and the capsular damage is less severe Shoulder dislocations, therefore, are seldom complicated by avascular necrosis of the bone Furthermore, fractures of the neck of the humerus seldom lie above all capsular attachments and the blood supply of the head of the humerus is usually preserved

When there is a fracture-dislocation of the joint the blood supply is more precarious If every soft part attachment to the head of the bone is torn, the bone will undergo necrosis whether it is reduced or not In one case a typical fracture-dislocation of the shoulder was manipulated and the reduction appeared to be complete.[2] Post-reduction radiographs showed that the head of the humerus was actually in the shoulder joint, but it was upside down and its fractured surface was in contact with the glenoid. At that time the significance of avascular necrosis was not recognised; an operation was performed and the head was tilted round into its proper position The reduction was perfect, but the loose head underwent complete necrosis and disintegration The unnatural mobility which allowed the fragment to lie upside down was sufficient evidence to prove its complete detachment from soft parts Avascular necrosis was therefore inevitable and the fragment might just as well have been left upside down Ankylosis in the optimal position of moderate abduction was the best possible result In such cases, if bony fixation does not occur spontaneously, arthrodesis is advisable.

[1] Maydl, K *Wien klin Rdsch*, 1897, **11**, 153, 171, 187
Legg, A T *Boston med Surg J*, 1910, **162**, 202
Calvé, J *Rev Chir*, *Paris*, 1910, **42**, 54
Perthes, G C *Dtsch Z Chir*, 1910, **107**, 111, *Arch klin Chir*, 1913, **101**, 779
Platt, H *Brit J Surg*, 1921, **9**, 366
Phemister, D B *J Bone Joint Surg*, 1930, **12**, 769
Danforth, Murray *J Bone Joint Surg*, 1934, **16**, 516.
Waldenstrom, H *J Bone Joint Surg*, 1938, **20**, 559
Waldenstrom, H "Die Tuberkulose des Collum femoris im Kindesalter und ihre Beziehungen zur Huftgelen-kentzündung" Stockholm P A Norstedt & Soner, 1910
Gill, Bruce *J Bone Joint Surg*, 1940, **22**, 1013
Jackson Burrows *Brit J Surg*, 1941, **29**, 23
[2] Watson-Jones, R "Fractures in the Region of the Shoulder Joint" *Proc Roy Soc Med* (Section of Orthopædics), 1936, **29**, 1070

AVASCULAR NECROSIS OF THE CARPAL SCAPHOID BONE

Blood supply of the scaphoid—On the evidence of radiographic examination of scaphoid bones after injecting the vessels with opaque media, it was formerly accepted that the arterial supply was from two main vessels, one entering the tubercle and one the waist [1] If this was correct, almost every fracture of the waist and certainly every fracture of the proximal pole should deprive the proximal fragment of the scaphoid of its blood We know from clinical observation that this is not so Avascular necrosis of the proximal fragment is seen only in a proportion of proximal pole fractures, and in a still smaller proportion of waist fractures



Fig 171

Fracture waist of scaphoid, the proximal fragment is completely avascular It is no less dense than the upper shafts of the radius and ulna whereas the other carpal bones are decalcified

Examination of a large series of scaphoid bones shows that the vascular foramina, which are situated in the ligamentous ridge between the two main articular surfaces, conform to two types of distribution [2] In two-thirds of the bones the vessels are distributed equally throughout the length of the ligamentous ridge (Fig 172) In the other third, there are no vessels directly entering the proximal half They pierce the cortex of the distal half and travel backwards in the bone The foramina may be confined to the tubercle, or there may be a few small, or one or two large, foramina actually at the waist These facts explain the unequal rate of union of fractures of the tubercle, the waist and the proximal pole of the scaphoid, and they explain the variable incidence of arthritis of the wrist-joint after these injuries

Fractures of the tubercle do not interfere with the blood supply of the fragments in either type of bone The fracture is in a vascular region and it should unite rapidly and with certainty We find that this is so. Fractures of the tubercle always unite within a few weeks and non-union is unknown

Fractures of the proximal pole, on the other hand, must deprive the proximal fragment of its blood if the distribution of vessels is of the second type, but not if it is of the first This again coincides with clinical experience Two-thirds of the fractures in this situation behave like fractures of the waist, and they unite promptly if immobilisation is adequate In one-third of the fractures, union is very slow It may be necessary to continue immobilisation for twelve months, or even longer, and if immobilisation is not prolonged the fracture does not unite

[1] Schnek, Fritz *Zbl Chir*, 1930, 57, 2600
[2] Obletz, B E, and Halbstein, B M "Fractures of the Carpal Navicular" *J Bone Joint Surg*, 1938, 20, 424



Fig. 172

The blood vessels of the scaphoid are usually distributed throughout the length of the bone, as in the specimen on the left, but in one-third of scaphoids all the blood vessels enter the distal half, as in the two specimens on the right

Fractures of the tubercle

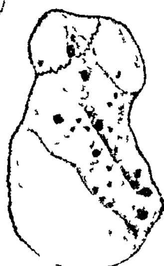


Fig 173



Fig 176



Fig. 179

Fractures of the waist.

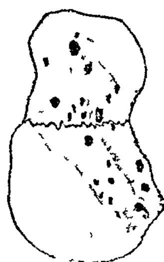


Fig 174

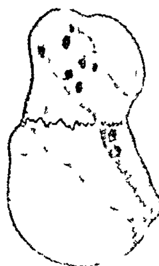


Fig. 177



Fig. 180

Fractures of the proximal pole



Fig 175



Fig 178

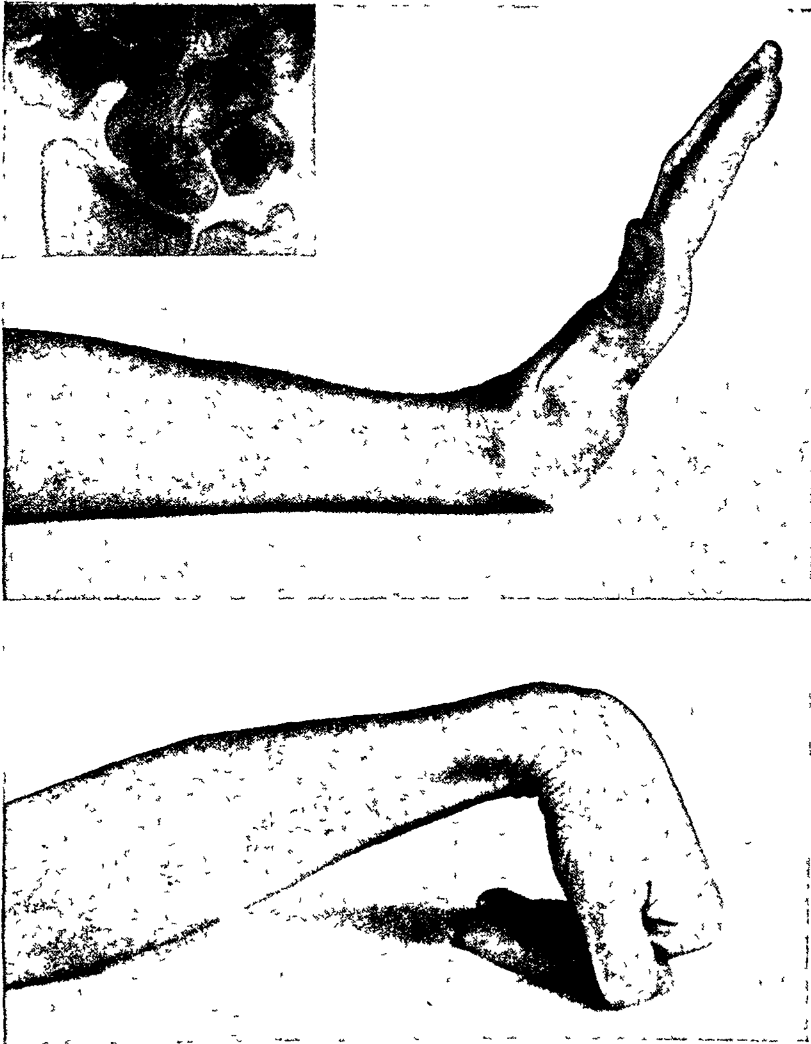


Fig 181

Figs 173-181

With the first type of blood supply, in which vessels enter the bone throughout its length (Figs 173-175), there is no danger of avascular necrosis from any fracture, it occurs only after dislocation With the second type of blood supply, necrosis of one fragment sometimes occurs in waist fractures (Figs 176-178), and always occurs in proximal pole fractures (Figs 179-181)

Fractures of the waist usually unite with certainty after six or eight weeks of complete immobilisation In two-thirds of the cases the blood supply of the fragments is not even in danger In the remaining third, however, the supply is precarious The fracture may lie proximal to all



Figs 182-184

Fracture of scaphoid with avascular necrosis of the proximal half, treated by early excision of the dead fragment There is perfect movement and strength, and complete freedom from pain.

vessels, or it may damage and thrombose the one large artery which enters the waist Union will then be slow exactly as in proximal pole fractures

Fracture-dislocation of the scaphoid—If there is not only a fracture of the waist but also a dislocation of the proximal fragment, there is a still greater likelihood of vascular impairment Whatever the type of blood supply, it is in danger of being cut off by dislocation Slow union is therefore more frequent after fracture of the scaphoid with dislocation of the lunate and half scaphoid than it is after simple fracture

Arthritis of the wrist after scaphoid fracture—We have seen that fractures can unite despite an avascular fragment if immobilisation is prolonged, sometimes for twelve months to two years (Figs 16-21). Slowness of union is not, however, the only penalty If the fracture is not immobilised long enough for sound union, the proximal fragment cannot revascularise and regenerate Degeneration of articular cartilage, arthritis of the joint, and



FIGS 185-187

Similar case to Figures 182-184 treated by prolonged immobilisation Despite firm union the wrist is very stiff and painful There is arthritis involving the wrist and still more the mid-carpal joint

severe stiffness, must supervene in succeeding years Unfortunately, even when prolonged immobilisation is rewarded by union of the fracture, arthritis may still occur because the new fibro-cartilage is imperfect It is therefore clear that whenever there is complete loss of blood supply to one fragment, this fragment at least should be excised , or if the surgeon prefers it he may excise the whole bone

Indications for excision of the scaphoid—If the proximal fragment is completely dislocated, or if in any fracture there is evidence after three or

four weeks of complete failure of decalcification of the proximal fragment, it should be excised When the operation is performed without delay, an almost normal range of movement and a wrist joint little short of perfect can be secured (Figs 182-184). The decision must be made within a few weeks of injury Delayed excision is worthless As months pass by, the articular cartilage of the radius and the capitate, opposite to the necrotic scaphoid fragment, undergo secondary attrition and degeneration. Excision at this late stage fails to prevent the arthritis, and indeed the trauma of operation may even precipitate the arthritis that it was designed to prevent

At one time non-union of the scaphoid was very common and late excision of one or both fragments was frequently performed The work of Bohler[1] showed that union could usually be secured Excision fell into disfavour There is no doubt, however, that the operation must once more take its place, not as an indiscriminate treatment but as an operation with well-defined indications Like other surgeons, I gave up excision for several years and pursued with enthusiasm the treatment of immobilisation alone, or of drilling and immobilisation The success so far as union is concerned was undoubted In a consecutive series of 100 scaphoid fractures there was bony union in 95 per cent of cases less than one month old, and in 90 per cent of fractures less than one year old[2] But follow-up examination after several years proves that good function depends not simply on bone union but much more on freedom from avascular necrosis Figures 185-187 show the extremely limited range of movement in a case where necrosis and arthritis supervened despite firm union of the fracture The result would have been much better if the fragment had been excised within a few weeks of injury, and the wrist not immobilised at all *The fragment must, however, be excised at once, and if both fragments are not excised it must be the dead half which is removed, not the living half!* Figure 188 illustrates a case in which the living distal half has been removed and the dead proximal half has been left in the joint



FIG 188

Avascular necrosis of proximal half of scaphoid after fracture of the waist. *The wrong half of the bone has been removed!*

AVASCULAR NECROSIS OF THE LUNATE BONE

Blood vessels reach the carpal lunate bone from the posterior and anterior ligaments, and they enter through foramina on the dorsal and palmar interarticular surfaces There are three types of dislocation of this bone, each interfering with the blood supply in varying degree

[1] Bohler, L "The Treatment of Fractures" Vienna W Maudrich, 1929
[2] Watson-Jones, R "Inadequate Immobilisation and Non-union of Fractures Fractures of Carpal Scaphoid Bone" *Brit med J*, 1934, 1, 936

Retrolunar dislocation of the capitate—In the first type, the capitate is displaced backwards out of the cup of the lunate, but the lunate itself remains normally attached to the radius by dorsal and palmar ligaments. The blood supply still reaches the bone through both ligaments and whether the dislocation is recent or old it should be reduced Excellent results are secured even if reduction is delayed (Fig 189)

Forward dislocation of the lunate—In the second type, the dorsal ligaments of the lunate are torn and the bone is tilted forwards, away from the radius and capitate (Fig 190) The intact anterior ligament carries a reduced blood supply If the dislocated bone is replaced promptly by manipulation it usually survives, and a perfect result with complete freedom from arthritis may be expected; but if the dislocation is several months old, and especially if the bone can be replaced only by open operation, the

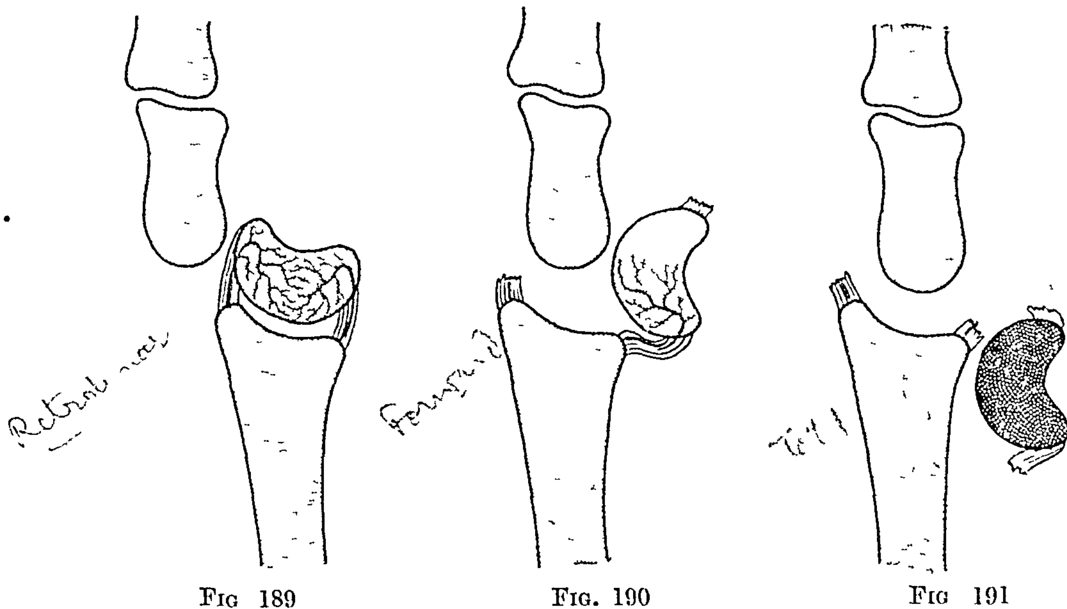


Fig 189 Fig. 190 Fig 191

There are three types of dislocation of the lunate. The blood supply is normal in the first, reduced in the second and cut off in the third

impairment of blood supply is increased, and although an excellent anatomical position may be secured, the functional result is usually imperfect. In a series of twelve old dislocations of the lunate of this type, which I reduced by operation some years ago, there is only one perfect result In all the others, wrist movement is restricted, and some are completely stiff. This was despite particular care and gentleness in operative reduction in view of the recognised danger of arthritis Dissection of soft parts was minimised, and a dorsal exposure was used in order to preserve the anterior ligament and the surviving blood vessels Nevertheless, avascular necrosis caused narrowing and arthritis of the mid-carpal joint between the lunate and capitate, and to a less extent of the radio-carpal joint These cases of old unreduced forward dislocation of the lunate are much better treated by excision

Total dislocation of the lunate—In the third type, both dorsal and palmar ligaments are completely ruptured and the bone, which is denuded of all soft part attachments, is displaced into the lower forearm (Fig 191). Avascular necrosis is then inevitable Traction and manipulation may

succeed in reducing the dislocation ; heroic methods of metacarpal skeletal traction with pins and wires have been described ; but successful reduction does the patient a disservice. The radiographic appearances are possibly more attractive than after excision , but the joint will become stiff, painful and susceptible to strain. Function is vastly superior after *early* excision Even if the patient is seen on the first day after injury, the widely displaced lunate which is obviously deprived of all its vessels should be excised.

AVASCULAR NECROSIS OF THE LATERAL CONDYLE OF THE HUMERUS

Children sometimes sustain a fracture of the lateral condyle of the humerus with tilting, rotation and wide displacement of the fragment. The common extensor origin of muscles, and the ligaments and capsule of the elbow joint, remain attached to the loose fragment and are responsible for its blood supply (Fig 192) If these tissues are dissected from the bone in the course of operative reduction, avascular necrosis will supervene. The fragment carries two-thirds of the articular surface of humerus, and very serious restriction of elbow movement remains Every attempt must be made to reduce these fractures by manipulation If manipulative reduction fails, operation should be undertaken with caution Difficult as the operation may be, the fragment must not be cleared of soft-part attachments Every muscular or capsular fibre arising from it is to be preserved, and the reduction must be carried out with the utmost care. If reduction is successful and the blood supply is not impaired the result will be perfect; whereas if the blood supply is destroyed and necrosis develops, there will be little or no movement below the right angle This disastrous result cannot be improved upon by excision of the fragment, because lateral instability, cubitus valgus deformity and delayed ulnar palsy will supervene It is therefore imperative that every effort should be made to preserve the blood supply of the fragment

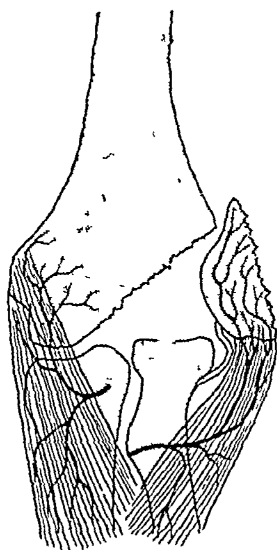


FIG. 192

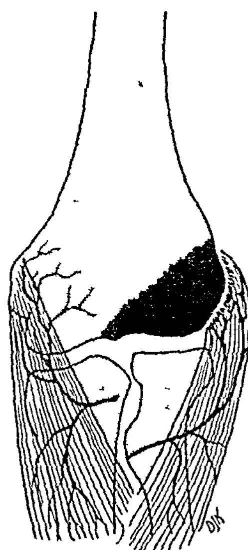


FIG 193

The displaced external condyle of the humerus depends for its blood on the capsule and muscles attached to it If these are dissected off during operative reduction, necrosis of the condyle and stiffness of the elbow joint supervene

AVASCULAR NECROSIS OF THE TALUS

Blood supply of the talus—The main blood supply of the talus is through the neck of the bone There are large foramina on its dorsal surface at the site of attachment of the anterior capsule of the ankle joint (Fig 194) Other vessels enter its ventral surface from the interosseous ligament in the sinus tarsi.

Fractures of the neck of the talus do not deprive either the head or the body of their blood supply (Fig 195) Even when this fracture is accompanied by dislocation of the subtaloid joint, so that all vessels from the sinus tarsi are destroyed, there may still be a sufficient blood supply from the dorsal and posterior vessels to both halves of the bone ; but when the fracture is complicated by backward dislocation of the body, all capsular attachments to the body of the bone are torn and it is entirely deprived of blood (Fig. 196) This half of the talus undergoes avascular necrosis. If



FIG 194

Blood supply of the talus Vessels enter the neck on dorsal and ventral surfaces, and also enter the body on its inner and posterior surfaces

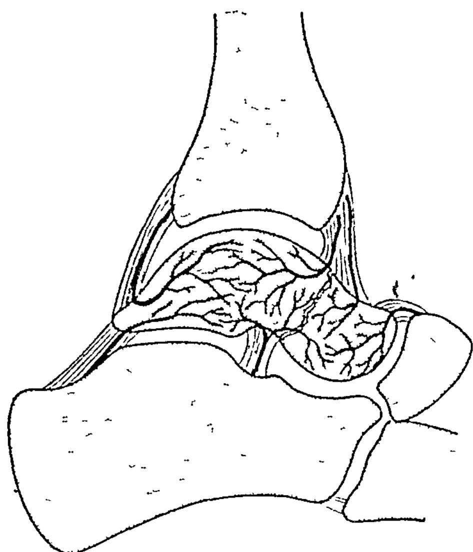


FIG 195

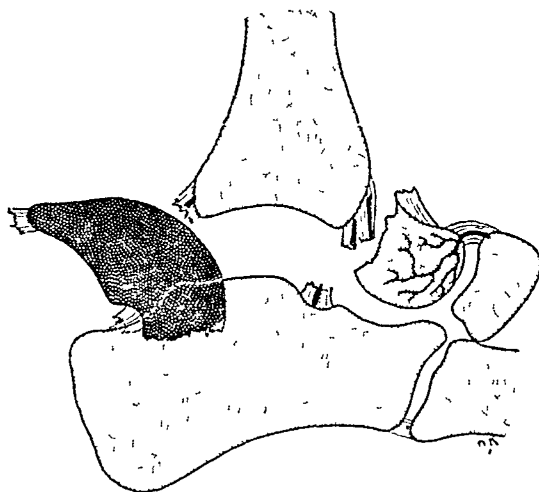


FIG 196

Fracture of the neck of the talus does not interfere with the blood supply (Fig. 195). If the body of the talus is also dislocated backwards, it undergoes necrosis (Fig. 196)

the displacement is reduced the fracture will unite and in the course of time the bone will regenerate (Figs 197-201). Protection in plaster must be continued and the resumption of weight-bearing deferred for many months in order to prevent crushing of the necrotic bone and degeneration of the articular cartilage If degenerative arthritis does supervene it is serious because both ankle and subtaloid joints are involved , and both joints must be arthrodesed.



FIG 197
Dislocation of the body of the talus



FIG. 198
Immediately after reduction.



FIG 199
Six weeks after reduction.

Figs. 197-201. Fracture neck of talus with dislocation of the body, avascular necrosis and gradual revascularisation

Fracture-dislocation of the talus with backward displacement and typical rotation of the body; the subtaloid articular surface faces outwards, and the ankle joint surface inwards (Fig 197) The dislocated fragment was replaced by manipulation (Fig 198). Six weeks later there is evidence that the body is avascular because it has not participated in the decalcification seen in neighbouring bones (Fig. 199). Immobilisation in plaster and protection from weight-bearing were continued for many months. Serial radiographs showed gradual revascularisation, proved by the developing



FIG. 200
Six months after reduction.

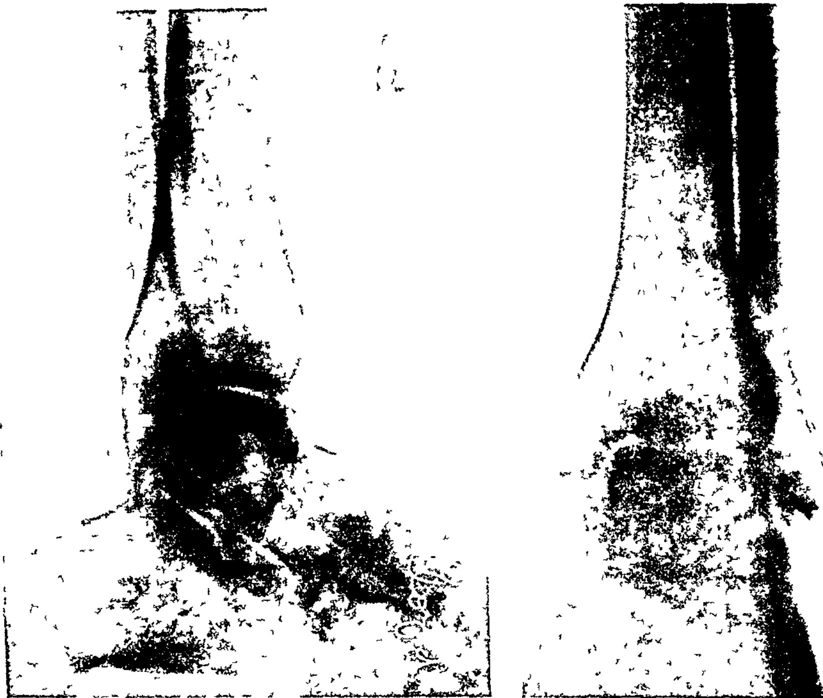


FIG 201
Twelve months after reduction.

of areas of decalcification, first in the region of the fracture and then spreading gradually backwards (Fig 200) The last area to revascularise was the subchondral bone and articular cartilage of the ankle joint Both ankle and subtaloid joints show some narrowing of the joint space, indicating thin articular cartilage (Fig 201).

In deciding that there is relative density of the body of the talus, care must be taken not to overlook the fact that even in the normal ankle the overlap of three shadows in the lateral radiograph—lateral malleolus, body of talus, and medial malleolus—gives an appearance of increased density

Arthrodesis of one of these joints does not cause serious disability because function is taken over by the other, and surprisingly free mobility remains; but if it is necessary to arthrodese both joints, the foot is rigid and inelastic. Nevertheless, despite the rigidity, the functional result of double arthrodesis is very much better than that of astragalectomy. After astragalectomy, inversion deformity nearly always develops, and the foot is weak and susceptible to strain.

Dislocation of the whole talus is an unusual injury which may be followed by the same complication A few soft-tissue attachments may remain to preserve a sufficient blood supply. Every effort should therefore be made to reduce the dislocation by simple manipulation, because operative reduction will almost certainly damage any vessels that remain.

AVASCULAR NECROSIS AND OSTEOCHONDRITIS DISSECANS

Necrosis of the articular surfaces in intra-articular fractures—Any intra-articular fracture that completely separates a fragment of bone and articular cartilage causes necrosis of the detached fragment. Small fragments of the capitellum may be detached by the impact of the head of the radius which itself sustains a marginal fracture. The capitellar injury is often overlooked, but there can be little doubt that avascular necrosis of the articular cartilage explains the limited elbow movement which is so common after fractures of the head of the radius These injuries must be regarded with suspicion. Good results are possible only if detached and loose fragments are removed at once Delayed excision of the radial head will not always relieve elbow stiffness because arthritis due to necrosis of the fragments is already developed. Similarly, fragments of the articular surfaces of the knee joint may be broken off and undergo necrosis. The lateral tuberosity of the tibia is sometimes comminuted by the impact of the lateral femoral condyle forced into the tuberosity by extreme valgus deformity. There is a large marginal fragment of the tuberosity which preserves its soft-tissue attachments and blood supply, and several smaller completely loose fragments lying between it and the main bone The cartilage of these fragments will necrose and it should not as a rule be elevated to the joint surface It is better to replace the main marginal fragment which is alive, and to leave a central crater of depressed dead fragments If the fragments are displaced between the joint surfaces they should be removed, not replaced, great care being taken to preserve all soft-tissue attachments to the marginal fragment of the tuberosity.

Osteochondritis dissecans—In other cases, fragments of the femoral condyles become separated and necrotic without a clear history of injury. This is the condition known as osteochondritis dissecans. The pathology is undoubtedly that of avascular necrosis, and it seems probable that there is usually a traumatic factor The lesion always occurs in those areas of the knee, elbow and ankle joints that are susceptible to trauma and where true fractures of the joint surface are often seen. In the knee joint it usually occurs on the medial surface of the lateral femoral condyle where it may be injured by impact against the tibial spine (Fig 202) The articular surface of the patella may be involved and is often damaged by contact with the femoral condyle. In the capitellum it always occurs in the area of impact

of the radial head In the ankle joint there may be separation of fragments from the body of the talus, especially in footballers, either from direct injury or from impact of the bone against the tibia In all these cases there is clear evidence of direct injury to the joint surface, usually from the impact of an articulating bone.

Osteochondritis dissecans at multiple sites—Sometimes, however there is not only no history of injury but the lesion occurs almost simultaneously at many sites in many joints One patient, for example, within the short period of two years, developed osteochondritis dissecans of the femoral condyle in one knee, the patella of the opposite knee the lateral condyle of one elbow, and the head of the radius in the other elbow Many adolescents have developed two, three or four lesions in both knee joints In such cases it is difficult to believe that there is not some general constitutional factor causing embolism or thrombosis of the end-arteries which supply these fragments of bone and articular cartilage



Fig 202

Osteochondritis of the lateral femoral condyle There is a well-defined crater from which the loose body separated as a necrotic fragment of bone and articular cartilage.

No such embolism has been proved, but there are of course many possibilities For example, we know that in general diseases where the sedimentation rate is raised, clumped or agglutinated masses of red corpuscles circulate and are capable of blocking end-arteries Similar clumping always occurs as a local response to injury, and microscopy of living tissue shows these masses being washed off and liberated into the general circulation It is not at all difficult to postulate embolism of end-arteries supplying local areas of joint surfaces in adolescents who are subject to intercurrent disease and who sustain many minor injuries.

CHAPTER VI

VASCULAR INJURIES

"*An amputation below the knee, in most cases, would not kill by its hæmorrhage even if left to itself*"—John Hunter

This courageous observation was made by Hunter 175 years ago after studying the contraction of the muscular coat of arteries in response to injury.[1] When he divided the blood vessels in the thigh of a boar, bleeding ceased "before the animal weakened" After exposure of the posterior tibial artery of a dog, the vessel "was observed to be so much contracted in a short time as almost to prevent the blood from passing through it, and when divided, the blood only oozed out from the orifice" Hunter's work was the first recognition of segmental spasm of arteries—the vasoconstriction that prevents rapid and complete exsanguination after severe wounds His observations have been confirmed by later clinical experience. Makins[2] recorded cases in the 1914-18 war of large arteries severed by bullets without external hæmorrhage or hæmatoma formation In the recent war, an airman's foot was severed at the ankle joint by the propeller blade of an aircraft; he was admitted to hospital in excellent condition having suffered little loss of blood A motor-cycle dispatch rider severed his leg two inches below the knee joint, and a bystander reported that he lost less than half a pint of blood, an improvised tourniquet consisting of a handkerchief and a twig was placed in position, ready to be tightened, but it was never needed; on admission to hospital his systolic blood pressure was 92 mm, the hæmatocrit reading was 52 per cent, and he made a good recovery Figure 203 shows a sailor who went ashore for the evening, drank well but not wisely, and then tried to find his way to the railway station One hour after the last train had passed he was found on the railway line with his upper limb severed at the elbow He had not bled to death; on the contrary he was admitted to hospital with a systolic blood pressure of 120 mm and a diastolic pressure of 85 mm, and he was discharged within two weeks Holdsworth reported two patients with "avulsion of the whole upper limb, including the clavicle and scapula, in whom there was no arterial bleeding"[3] These cases could be multiplied They illustrate the fundamental principle that arteries respond to injury by "traumatic arterial spasm"

THE TYPES OF ARTERIAL INJURY

Traumatic arterial spasm—Spasm of the muscular coat of an artery occurs not only after severance and rupture but also after contusion or even "concussion" of the vessel The disruptive force of a bullet, traversing the tissues near an artery but not actually producing a demonstrable lesion of the vessel, may cause segmentary spasm, *stupeur artérielle*, or Kroh's arterial spasm[4] as it was called by English, French[5,6] and German observers

[1] "Works of John Hunter" Edited by Palmer London Longman, 1835, **1**, 538, and **3**, 157
[2] Makins, G "On Gunshot Injuries to the Blood-Vessels" Bristol John Wright & Sons, 1919
[3] Holdsworth, F W *Med Pr*, 1942, **208**, 300
[4] Kroh, F *Beitr klin Chir*, 1915, **97**, 345, and 1917, **108**, 61
[5] Ducastaing, R *Bull Mém Soc Chir*, *Paris*, 1919, **45**, 604
[6] Soubeyran, P, and Michon, E *Bull Mém Soc Chir*, *Paris*, 1918, **44**, 805

in the last war. Kroh described a revolver bullet wound in the region of the femoral artery which arrested the circulation of the limb; at operation the artery was found apparently undamaged but contracted to the size of a knitting needle, to the surgeon's surprise "while the artery was exposed it dilated to normal size." Cohen[1] records a recent case of a penetrating wound of the elbow by a bomb splinter, with absent radial and ulnar pulses, the brachial artery was so contracted as to be smaller than adjacent venæ comites, although actual injury was limited to fine stippling of its wall;



Fig. 203

Sailor who lay on a railway track for one hour after his forearm had been severed He did not bleed to death Hæmorrhage was controlled by vasospasm

simple exposure of the artery and separation from its bed of areolar tissue was enough to relieve the spasm.

Traumatic arterial spasm may also occur when there is no external wound. The artery may be contused by one of the fragments of a fractured bone, and this is now recognised with increasing frequency as a cause of ischæmic contracture and gangrene The brachial artery is threatened in supracondylar fractures of the humerus, the radial and ulnar arteries in fractures of the forearm, the axillary artery in fractures of the neck of the humerus, and the popliteal artery in high oblique fractures of the tibia. A violent blow that does not fracture the bone may cause traumatic arterial spasm and gangrene, as for example in "bumper injuries" of the knee where the tibial artery is contused against the neck of the fibula[2] Counterpressure on the popliteal vessels by the cross-bar of a Bohler's traction frame may be responsible[3] Similarly, the sharp pressure of a ridge of plaster may traumatise an artery Trueta[4] reported the case of a patient whose fractured elbow was immobilised in plaster in extension, a few moments later an assistant walked in "and believing that a mistake had been

[1] Cohen, S *Lancet*, 1941, 2, 806, and *Guy's Hosp Rep*, 1940, 90, 201.
[2] Jones, S G *Amer J Surg*, 1939, 43, 325
[3] Cohen, S M *Guy's Hosp Rep*, 1949, 90, 211
[4] Trueta, J *An Hosp S Cruz S Pablo, Barcelona*, 1934, 8, 198

made, proceeded to flex the joint before the plaster had set," thus buckling the cast in the fold of the elbow The circulation was completely arrested and at operation the brachial artery was found to be reduced to one-third



Fig. 204
Normal arteriogram.



Fig. 205
Arteriogram in ischæmic contracture

Vascular occlusion from fracture of the forearm

Arteriograms after injection of perabrodil in a case of shrapnel wounds without vascular injury (Fig 204) and of ischemic contracture after fractures of both forearm bones (Fig 205). The radial artery has been contused by the fracture of the radius, and the interosseous artery by the fracture of the ulna, the ulnar artery is intact.

its normal size Many cases have been recorded of the tight application of tourniquets causing arterial spasm which persisted long after the tourniquet had been released In one case the femoral artery remained pulseless eighteen hours after removing a tourniquet which had been left on the limb for six hours.[1] Exposure of the artery showed no hæmatoma and

[1] Griffiths, D. Ll. *Brit. J. Surg.*, 1940, 28, 256.

no arterial laceration but only spasm, which was relieved by division of the fascial roof of Hunter's canal and mobilisation of the artery. Esmarch introduced his flat rubber tourniquet sixty-five years ago because gangrene had followed the use of inelastic tourniquets, but even the Esmarch tourniquet is not safe. Vasospasm of the brachial artery causing gangrene of the forearm and hand has occurred from the pressure of an Esmarch rubber tourniquet at the lower axillary margin for only forty-five minutes (Fig. 227). The only safe tourniquet is the pneumatic tourniquet, with a sustained and controlled pressure of 300 mm.

The use of rigid tourniquets may be responsible for not only local vasospasm and gangrene but also vasospasm of the opposite limb and even renal changes leading to uræmia and death.—Spasm of the artery is not limited to that part of the vessel sustaining injury; it involves also the main branches, the vessels of the collateral circulation, and sometimes the trunk of the artery proximal to the level of injury. Clearly, therefore, it is more than a local myogenic reaction of injured tissue in the wall of an artery.[1,2] It is a reflex vasoconstriction, the afferent impulses arising from the damaged region of the artery, passing to the paravertebral plexus, and stimulating the whole sympathetic nerve supply of the affected limb.[3] Recent evidence shows that it may overflow to the opposite limb and cause vasospasm in the main artery on the uninjured side. Moreover, in the case of lower limb injuries, vasospasm may extend as high as the renal vessels, thus accounting for the uræmia that complicates crushing injuries of the lower limbs—the so-called "crush syndrome."[4,5] The reflex character of the spasm is suggested not only by its widespread distribution, but also by the relief that is usually gained after removal of the source of afferent impulses by resection of the injured segment of artery[6] or by cutting the sympathetic reflex arc by paravertebral novocaine injection,[7] spinal or brachial anæsthesia, or operative sympathectomy.[8] A dramatic case illustrating these features of traumatic arterial spasm—the minor degree of injury, the widespread distribution of spasm and the favourable response to excision of a segment of the vessel—was reported by Griffiths.[9] A boy aged seven years sustained a supracondylar fracture of the humerus. "Before reduction of the fracture the hand and forearm were normal, but immediately after the manipulation they changed dramatically, becoming suddenly blanched, cold, pulseless and anæsthetic. I explored the arm and found a small hæmatoma no larger than a millet seed in the wall of the brachial artery at the site of fracture. Despite the smallness of this hæmatoma the whole artery and its radial and ulnar branches were collapsed and pulseless from the insertion of the coracobrachialis downwards. The collateral vessels were equally collapsed—the anterior ulnar recurrent was the size of a retinal artery. Mobilising and stripping the brachial artery produced no change, but after resection of the damaged portion the arm made a complete recovery. Circulation was fully restored, and there was neither gangrene nor contracture."

[1] Kuttner, H., and Baruch, M. *Beitr. klin. Chir.*, 1920, **120**, 1.
[2] MacWilliam, J. A. *Proc. Roy. Soc. Med.*, 1902, **70**, 109.
[3] Leriche, R. *Ann. Surg.*, 1928, **88**, 449.
[4] Barnes, J. M., and Trueta, J. *Brit. J. Surg.*, 1942, **30**, 74.
[5] Trueta *et al.* "Studies of the Renal Circulation." Oxford: Blackwell Publications Ltd., 1947.
[6] Leriche, R., and Werquin, M. G. *Lancet*, 1940, 2, 296.
[7] White, J. C. "The Autonomic Nervous System." New York: Macmillan Co., 1935.
[8] Gage, M., and Ochsner, A. *Ann. Surg.*, 1940, **112**, 938.
[9] Griffiths, D. Ll. *Brit. J. Surg.*, 1940, **28**, 239.

The underlying purpose of reflex vasospasm is protection from fatal bleeding after severe arterial injury; but, for this protection, the animal must pay the price of ischæmic contracture or even gangrene. The spasm usually persists for at least twenty-four hours, and sometimes for three or four days. The distal pulse may then return, but it is too late; the damage has been done; the muscles are necrosed and, despite the return of a strong pulse, the limb may still become gangrenous. Treatment is therefore urgent. If grave complications are to be avoided the circulation must be restored within about six hours. Non-operative measures may be tried, including intravenous injection of antispasmodics,[1] paravertebral novocaine injection of the sympathetic ganglia,[2] or interruption of the vasoconstrictor fibres by brachial plexus block or spinal anæsthesia. The most certain and reliable treatment is early exposure of the artery, separation from its bed, and removal of the surrounding areolar tissue. Hot saline lavage of the wound or massage of the artery may assist. If spasm still persists, arteriectomy may be unavoidable, particularly when the wall of the artery shows bruising. Arteriectomy should, however, be the very last resort; every other effort should first be made to overcome the spasm.

Traumatic venous spasm—It is to be noted that vasospasm is not limited to the arteries but may also involve the veins. Every surgeon who has ligated the saphenous vein for varicosity has had the opportunity of observing rapid contraction of the vein when it is handled. In thrombophlebitis, constriction of the veins occurs in the collateral venous network and gives rise to œdema which could not be explained by simple occlusion of one vein. Moreover, thrombophlebitis causes arterial spasm which disappears on novocaine infiltration of the lumbar sympathetic.

Arterial contusion, thrombosis and embolism—Contusion of an artery may obstruct the lumen of the vessel by causing thrombosis at the site of a crack in the tunica intima. The circulation is often maintained by the aid of collateral vessels, but sometimes it is further obstructed by distal extension of the thrombus, or by the release of an embolus which impacts at a lower level at the opening of a large branch, or at a site of bifurcation. Moreover, the irritant effect of a thrombus or embolus within an artery is itself sufficient to induce secondary arterial spasm and cause further embarrassment to the circulation. Secondary vasospasm may indeed be so widespread as to confuse the clinical signs and create difficulty in establishing the level of obstruction. For example, in one case, a small embolus lodging in the posterior tibial artery gave rise to reflex vasospasm involving both legs, thus resembling the clinical picture of obstruction at the bifurcation of the aorta.[3] The cumulative effect of thrombosis, embolism and reflex vasospasm leading ultimately to gangrene, is illustrated in Figures 206-208, a case of un-united fracture of the clavicle with repeated

[1] Papaverine hydrochloride (½ gr.) is injected intravenously. If vasodilatation does not occur, and there are *no untoward effects, a second dose of ½ gr.* may be given in thirty minutes. Kvale, W., and Allen, E. V., Collected Papers, Mayo Clinic, 1936, 28, 617.

[2] Injection of the sympathetic ganglia with 2 per cent. novocaine at the 2nd and 3rd thoracic intervertebral spaces for the upper limb, and the 2nd, 3rd and 4th lumbar spaces for the lower limb, is strongly advocated by J. C. White ("The Autonomic Nervous System." New York: MacMillan Co., 1935). Needles 10 cm. in length are inserted through the skin 3 cm. lateral to the upper margin of each spinous process, pushed perpendicularly inwards to a depth of 4 to 5 cm. until they touch the transverse process, and then directed slightly upwards and inwards and thrust 4 cm. through the psoas until the side of the vertebral body is felt. In order to exclude the dangers of injecting into an artery or the subarachnoid space the needles should be inserted independently, the syringe only fitted to them after they are in position, and aspiration attempted before novocaine is injected. Ten cubic centimetres of 2 per cent. novocaine is then injected at each level. Rapid warming and drying of the feet proves that the needles have been accurately placed.

[3] [illegible] J. S., *Surg. Gynec. Obstet.*, 1942, 74, [illegible]



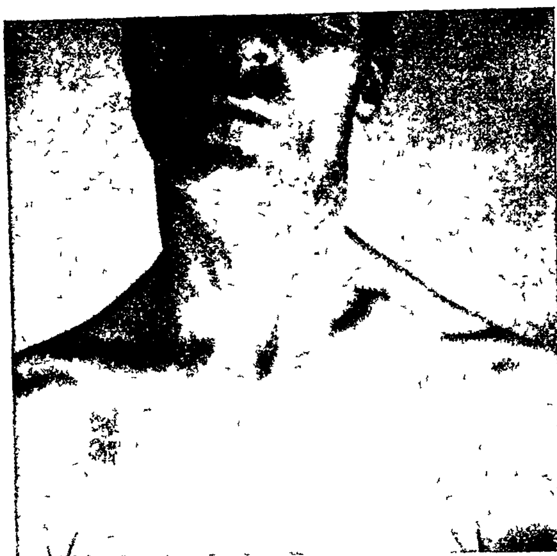


Fig 207

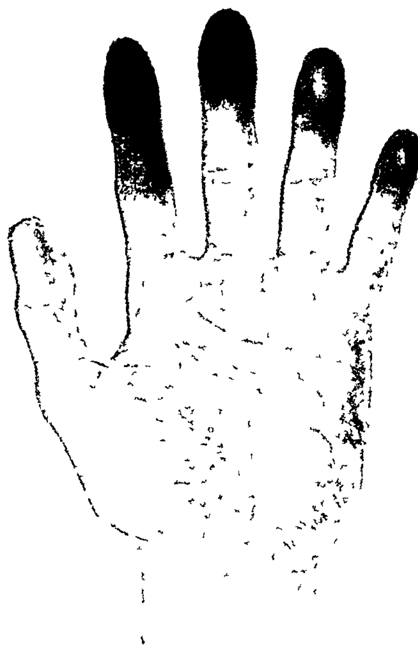


Fig 208

Fig 206

Arterial contusion causing embolism and vasospasm

Un-united fracture of clavicle with repeated contusion of the subclavian artery by the outer fragment causing thrombosis, embolism and vasospasm At the site of contusion (top of the specimen) there is roughening of the tunica intima, emboli detached from this site have blocked both main branches. Continued contusion of the periarterial sympathetic nerves caused vasoconstriction of collateral vessels, and finally gangrene of two fingers

artery are retracted and out of sight, it is still essential to find them and apply ligatures

Secondary hæmorrhage—Secondary hæmorrhage in infected wounds must be controlled by exploring the wound and finding the bleeding vessel, and not by ligation of the main artery at a more proximal level Proximal ligation of the main artery often fails to control the bleeding, it adds the risk of gangrene of the limb and, by reducing oxygenation of the tissues, it lowers the defence of the wound to infection, particularly anaerobic infection. The surgeon may be faced with great difficulties Hæmorrhage is sometimes so brisk that he cannot see, the wound fills with blood faster than a dab can be withdrawn, he is tempted to plunge hæmostat forceps into the bloody pool and clamp blindly whatever comes within reach. One young resident surgical officer, far exceeding his duty in attempting operative reduction of a late unreduced dislocation of the shoulder, "divided a strong adhesion" Before bleeding from a severed axillary artery had been controlled, all trunks of the brachial plexus had been crushed irreparably. The solution to the problem of finding the bleeding point in a rapidly filling pool of blood is the use of many gauze packs, firmly pressed into the wound *and left undisturbed for several minutes*. The incident recorded in the footnote below[1] illustrates the relative ease with which forceps can then be applied

Dangers of ligation in continuity—When division of an artery is incomplete, the vessel must not be ligated in continuity Ligatures should be applied above and below the level of perforation, the injured segment then being excised This is necessary not only to avoid reflex vasospasm, but also to prevent later erosion of the vessel wall When a large artery is ligated in continuity the constant battering of pulse beat is applied to a fixed point, and late erosion with secondary hæmorrhage is always a danger, on the other hand, section of the vessel allows elastic retraction and dissemination of the force of the pulse beat

Simultaneous ligation of vein—The danger of gangrene after ligature of the main arteries of the limbs is greatest in the case of the common femoral and the popliteal arteries The risk is less when the accompanying vein is ligatured simultaneously This has been confirmed by experimental evidence,[2] by clinical studies,[3,4] and by the report in 1917 of the Inter-Allied Conference of Surgeons in Paris which recommended "that the ligation of a large artery for injury should be accompanied also by occlusion of the satellite vein even although the latter be uninjured" The exception to this rule applies in the case of the "Henle Coenen phenomenon"[5], if after ligation and division of an artery the distal stump is seen to pulsate, it is clear that

[1] A surgeon in France, visited by members of a travelling surgical club, was performing a nephrectomy One of his visitors recalled the surgeon's reputation for dealing successfully with the problem of the ligature slipping off the renal pedicle What was the secret? "I will show you," was the reply, as he cut off the kidney with a pair of scissors, without first applying either forceps or ligature to the renal pedicle, artery or vein "There is the problem," he said, stuffing large gauze packs tightly into the cavity He left the operating table, commented on the weather, discussed the political situation and drank a cup of tea Five minutes passed before he returned to the task On gently removing the packs, the field remained dry quite long enough for him to apply hæmostat forceps with great deliberation to both artery and vein His secret lay in the exercise of restraint, and in allowing sufficient lapse of time for the hæmorrhage to be controlled by sustained pressure and reflex vasospasm

[2] Reichert, F L *Johns Hopk Hosp Bull*, 1931, **49**, 86

[3] Sehrt, E *Med Klin Berl*, 1916, **12**, 1338 A review showing gangrene in 20 per cent of lower limb ligations of artery alone, and only in 9 per cent of ligation of artery and vein, and in the upper limb, gangrene in 7 8 per cent of ligations of artery alone and in no cases of ligation of artery and vein

[4] Heidrich, L *Beitr Klin Chir*, 1921 **124**, 607 A review of 995 ligations of large arteries alone with gangrene in 154 (15 5 per cent) and of 198 ligations of both artery and vein with gangrene in 17 (8 5 per cent)

[5] Pemberton, J, and McCaughan, J *Ann Surg*, 1932, **96**, 1103

the collateral circulation is already adequate, and simultaneous ligation of the accompanying vein is then unnecessary.

Suture of artery—Successful suture of ruptured blood vessels has become possible since the introduction of heparin by which thrombosis can be controlled Heparin in saline is injected into the repaired vessel proximal to the line of suture and continued after operation by intravenous infusion for four or five days[1,2] End-to-end anastomosis is performed with fine silk on eyeless needles, using a continuous vertical mattress stitch which everts and approximates the edges and exposes a minimum of thread to the lumen of the vessel The arterial wound should be moistened from time to time throughout the operation with sterile sodium citrate solution Longitudinal wounds may be sutured in the long axis of the vessel, but if there is a transverse wound exceeding one-third of the circumference the artery should be divided completely and repaired by end-to-end anastomosis

Temporary arterial bridging with vitallium tubes[3]—A shouldered tube of vitallium, ligated in each end of the severed vessel, may be used as a temporary bridge until a collateral circulation is established The tube is of smaller calibre than the artery Heparin is given continuously. After about five days the heparin is stopped Twenty-four hours later the tube is removed and the vessel is ligated proximally and distally

Permanent arterial bridging—Attempts have been made to achieve a permanent arterial bridge by means of a vitallium cannula lined by a free vein graft from the saphenous vein Free vein grafts, inserted in reverse so that the valves do not obstruct circulation, have also been used without a vitallium tube. These methods are still on trial

Pulsating hæmatoma and traumatic aneurism—If a perforated artery does not communicate with an open wound, bleeding into surrounding tissues gives rise to an arterial hæmatoma After several weeks or months the hæmatoma resolves, pulsation develops, and a traumatic aneurism is formed Rupture of an artery is occasionally due to simple contusion A surgeon who was playing cricket stopped a fast ball in the palm of his hand. After two hours there was tingling, followed by intense pain in the distribution of the median nerve. The lightest touch on the thumb, index or middle fingers, or even a breath of air, was sufficient to precipitate agonising waves of pain. The hollow of the palm was filled and the front of the wrist tightly swollen Aspiration of 16 c c of blood gave temporary relief, but within twenty-four hours paralysis of the median nerve was complete Operation disclosed rupture of the superficial palmar arch with an arterial hæmatoma filling the space beneath the annular ligament and infiltrating the lumbrical muscle bellies. Division of the anterior carpal ligament gave immediate relief from pain, but several months elapsed before the median paralysis recovered[4]

Perforation of an artery may also be due to the penetration of a sharp spicule of bone Figures 209-211 show a comminuted Colles' fracture of the radius complicated by perforation of the radial artery and a traumatic aneurism which was first recognised several weeks after the plaster had been removed The femoral artery is sometimes perforated in the upper thigh

[1] Best C H *Brit med J*, 1948, 2, 977 From 20 to 100 mg of heparin in 20 c c of saline is injected into the vessel, and an intravenous drip containing 10 mg heparin per 100 c c saline is continued after operation at the rate of 30 drops a minute
[2] Murray, G *Lancet* 1939, 2, 133
[3] Learmonth J R *Lancet* 1944, 2, 745
[4] Watson-Jones, R "Carpal Tunnel Compression of the Median Nerve" *J. Bone Joint Surg*, 1949, 31-B, 560

by one fragment of a fractured shaft of the femur. The circumference of the thigh is increased several inches by a deeply fixed swelling, which is extremely hard, may slowly increase in size, does not at first pulsate, and may almost present the clinical picture of a sarcoma of bone. After several months as the hæmatoma undergoes resolution, the development of expansile pulsation and a systolic bruit make the diagnosis clear Regular

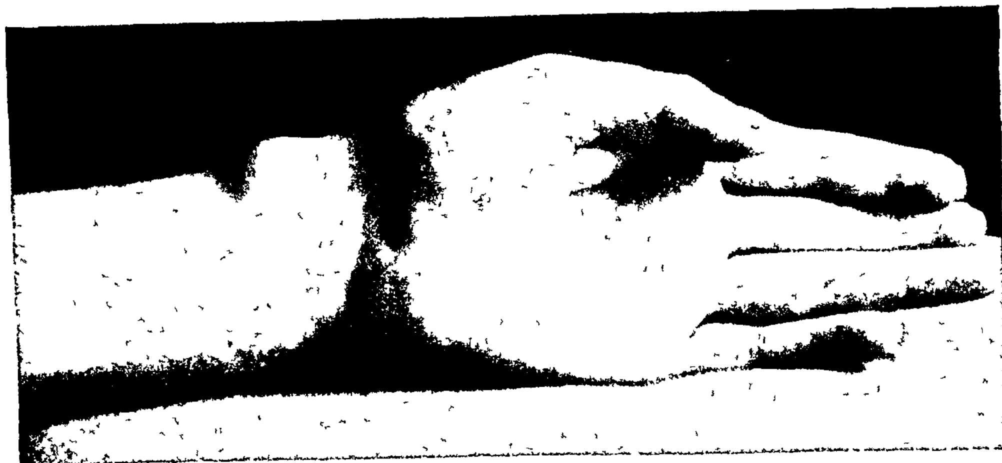


FIG. 209

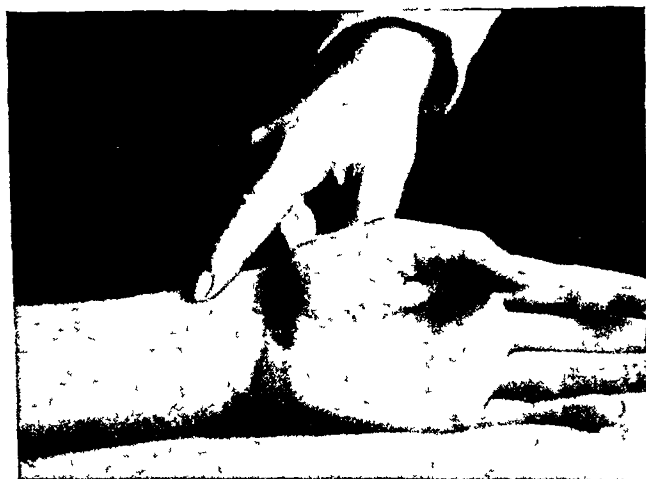


FIG 210



FIG 211

Traumatic aneurism

Aneurism of the radial artery due to puncture of the vessel by a bone spicule from a comminuted Colles' fracture

measurements of the circumference of the limb should be recorded If the hæmatoma is not increasing, and there is no threat of ischæmia or gangrene, operation should be deferred long enough to assist the establishment of a collateral circulation and to reduce the difficulties and danger of operation A delay of two or three months is advisable Whenever possible, the usual Hunterian ligation of the artery proximal to the aneurism should be supplemented by distal ligation and excision of the intervening segment. Before the operation is performed, special care must be taken to exclude the possibility of venous as well as arterial communication with the hæmatoma

If a venous communication is overlooked and an arteriovenous fistula is treated by proximal ligation of the artery alone, the result is disastrous

Arteriovenous fistula—Simultaneous perforation of artery and vein gives rise to an arteriovenous fistula, the communication may be direct (aneurismal varix), or through an intervening false aneurism (varicose aneurism) The diagnosis is established by the development of a thrill and bruit which is continuous throughout the cardiac cycle Continued escape of arterial blood into the veins and the right side of the heart calls for a greatly increased cardiac output in the attempt to maintain normal arterial pressure. If the fistula is large, more than half the circulating blood may leak back into the capacious venous bed Dyspnœa and tachycardia arise on the slightest exertion, attacks of momentary faintness develop, there is increasing "pounding" of the heart, slowly developing decompensation due to cardiac dilatation, and ultimately invalidism and death Occasionally there are local manifestations in the area of venous back pressure The veins may become enormously dilated, and there is varicose eczema and ulceration

The differential diagnosis between simple traumatic aneurism and arteriovenous aneurism is of the greatest importance, because whereas proximal ligation of the artery may suffice for a simple aneurism, this operation fails to cure an arteriovenous aneurism or to relieve the cardiac distress, and it usually causes gangrene of the limb Blood finds its way through the collateral circulation of the ligated artery back to the fistula and vein, and to the limb beyond the fistula with even greater difficulty than before. The distinctive features of a fistula as compared with an aneurism are (i) the thrill and bruit are continuous but intensified in systole in the fistula, and occur only in systole in the aneurism, (ii) digital closure of the artery proximal to the lesion slows the pulse rate and increases the blood pressure in the presence of fistula, but never in the presence of simple arterial aneurism

Early operation, soon after injury, is needed only when hæmorrhage from the wound continues or when an increasing hæmatoma threatens to obstruct the circulation of the limb The wound is then laid open, blood clot is removed, débridement is performed, and both artery and vein are ligated and divided Free drainage is established by leaving the wound open As a rule these aneurisms develop gradually, and several weeks elapse before they are clinically obvious In these circumstances, provided that cardiac decompensation is not threatening, operation should be deferred until at least three months after injury. Such delay assists the development of a collateral circulation and makes the operation easier and more safe. Complete rest in bed for two weeks before operation is advisable Digital closure of the fistula for thirty minutes, three to six times a day, may also assist in controlling cardiac decompensation The operation of choice is ligation of the artery and vein, proximal and distal to the fistula, with excision of the fistula [1]

THE RESULTS OF ARTERIAL INJURY

The types of arterial injury that have been considered include contusion of an artery causing traumatic arterial spasm, contusion of an artery causing thrombosis at the level of injury or embolism at a more distal

[1] Holman, E "War Injuries to Arteries and Treatment" *Surg Gynec Obstet*, 1942, 75, 183

level, perforation or rupture of an artery causing external hæmorrhage, perforation or rupture of an artery causing internal hæmorrhage, arterial hæmatoma and traumatic aneurism or arteriovenous fistula. Any of these injuries may imperil the circulation of the limb. Sometimes a collateral circulation is established and recovery is complete; on other occasions Volkmann's ischæmic contracture supervenes, and sometimes the limb becomes gangrenous.

Recovery, ischæmic contracture or gangrene?—In what circumstances is arterial injury followed by recovery, by ischæmic contracture or by gangrene? The vascular demands of the tissues of a limb are not uniform. The greatest susceptibility to ischæmia is shown by sensory and muscle nerve endings which lose their power of conduction within fifteen or thirty minutes. For this reason loss of the blood supply of a limb causes paralysis and anæsthesia even when the nerves are uninjured. The next most susceptible tissue is muscle which normally has a blood supply more vigorous than that of bone, skin or ligament. If ischæmia persists for six or eight hours, muscle dies, whereas skin can survive as long as twenty-four hours. Not only does muscle suffer more quickly than other tissues but its powers of regeneration are more limited. Unlike bone, where an avascular sequestrum may be revascularised and replaced by living bone, a muscle sequestrum is never completely replaced by living muscle, but only by fibrous tissue which undergoes contraction.[1] The fate of the tissues of a limb after vascular occlusion depends therefore upon the speed with which the circulation is restored by reopening original channels or by the development of collateral channels, and the extent to which the relative demands of tissues have been met in the interval. If a free and vigorous circulation is established within about six hours, recovery is complete. If the occlusion lasts more than six hours and the collateral circulation is not adequate, tissues with the highest vascular demands suffer most; muscles undergo necrosis and Volkmann's ischæmic contracture supervenes. If the collateral circulation is totally inadequate, no tissue survives and gangrene develops.

The collateral circulation—The adequacy of the collateral circulation depends upon three factors: (i) the age and constitution of the patient; (ii) the local pressure of extravasated blood, splints and plaster, or body-weight; (iii) the degree of reflex vasospasm arising from the damaged segment of artery.

Age and constitution—Maximal vasodilatation of blood vessels is possible in the young and, with advancing years, much of the resilience of the arterial tree is lost. The elasticity of vessels is reduced by degeneration and calcification and by the arteriosclerosis of diabetes, syphilis or alcoholism. Shock, collapse and hæmorrhage, by reducing blood volume and pressure, and causing general circulatory embarrassment, are important. The general condition of the patient often determines the onset of gangrene after vascular damage[2] and blood transfusion or oxygen therapy may be essential. Vasodilatation should be encouraged by hot drinks, by the application of heat to uninjured limbs (but not to the injured limb) and by alcohol. Smoking, which causes powerful vasoconstriction, should not be permitted.

Local pressure on collateral vessels—If the circulation of a limb is in doubt

[1] Nevertheless it has been shown by Rowland Hughes (*J. Bone Joint Surg.*, 1948, **30-B**, 586) that limited regeneration of ischæmic necrotic muscle is possible even in the human being.
[2] Makins, G. "Gunshot Injuries to the Blood Vessels." Bristol: John Wright & Sons Ltd., 1919.

it must not be enclosed in a complete plaster cast Swelling of an injured limb, continuing after the application of an unpadded plaster, may obstruct even a normal circulation and cause gangrene When the main artery has been damaged, and survival depends upon the collateral circulation, it is still more important that every source of external pressure should be removed. The limb may be supported on a cradle splint, if there is a fracture, light traction may be employed and a plaster slab is sometimes necessary, but every encircling bandage, strapping or plaster cast must be divided Even the pressure of body-weight will sometimes obliterate the field of collateral circulation After ligation of the external iliac or common femoral arteries, survival of the limb depends upon collateral vessels developing in the gluteal region If the patient lies on his back, constant pressure on the buttock obstructs the circulation, he should be nursed on his face If an artery is injured in a wound which becomes infected, the pressure of inflammatory exudates and œdema may obstruct the collateral circulation Free drainage is essential, the deep fascia must be divided in order that inflammatory swelling of muscles shall not give rise to constriction, no layer of the wound should be sutured. Even extravasated blood may cause such pressure on surrounding tissues as to reduce or obstruct the blood flow through collateral vessels For this reason gangrene occurs more often when arteries are perforated by missiles than when they are ligated by surgeons Statistics of 1914-18 showed that surgical ligation of the subclavian artery never caused gangrene, whereas perforating wounds of the subclavian artery caused gangrene in 9 per cent of cases [1] Gangrene occurred in 35 per cent of wounds of the popliteal artery and only in 26 per cent of ligations of the artery

Reflex vasospasm—The influence of reflex traumatic arterial spasm on the collateral circulation has been discussed in the opening pages of this chapter. Injury to the wall of an artery, or thrombosis within the artery, sets up reflex vasoconstriction which affects not only the vessel itself but all the arteries of the limb including those upon which the collateral circulation depends The vasoconstriction must be relieved by novocaine injection of the ganglionated sympathetic chain, the brachial plexus or the lumbar nerves and, if this is not successful, by exposure and mobilisation of the artery or even resection of the injured segment

"The limb on ice" [2]—A limb threatened with ischæmia and possible gangrene should not be heated, because this increases the oxygen demand, hastens the onset of gangrene and favours the growth of pathogenic organisms It has been recommended that the limb should actually be frozen by packing with ice, or immersion in ice water, because refrigeration inhibits bacterial growth and slows the local metabolism These advantages are offset, however, by reduced dissociation of oxygen from oxyhæmoglobin The limb in which the circulation is precarious should be at room temperature, or perhaps slightly cooled by means of an electric fan But the rest of the body should be warmed because this causes general relaxation of vasoconstrictor tone, not only in the regions that are warmed but in the whole body, and therefore in the affected limb If vasoconstrictor tone can be released the blood flow in muscles is more than doubled [3]

1 "History of the Great War (Medical Services)" London H M S O, 1922, 2, 170
2 *Lancet* 1941, 2, 734
3 Barcroft, H, and Edholm, O "Sympathetic Control of Blood Vessels of Human Skeletal Muscle" *Lancet*, 1946, 2, 513

If gangrene has already set in, but for any reason amputation must be deferred, a tourniquet may be applied and refrigeration used Pain is then relieved, the foul odour disappears and the general condition of the patient is improved A frozen limb can be amputated with no other anæsthetic; refrigeration inhibits the nerve endings, prevents pain and minimises shock. This, however, is the only indication for refrigeration It is certainly not indicated when it is hoped that the limb may survive.

VOLKMANN'S ISCHÆMIC CONTRACTURE

Until very recently it was believed that Volkmann's ischæmic contracture was the consequence of venous obstruction from tight splints, bandages or plaster used in the treatment of elbow fractures. The belief that it was almost invariably attributable to faulty treatment led to the award of large sums in damages against medical men who were held responsible. The theory of venous obstruction was based on the work of Brooks who ligated the veins of the sartorius muscle in dogs, and of Jepson who ligated the femoral veins [1-3] Recent operative experience makes it clear, however, that the lesions they produced experimentally were not the lesions we now find in human subjects Volkmann [4] himself, writing sixty years ago, believed that the contracture was due to "a continuous stoppage of the arterial blood," and this is now well established. It is known that whereas total arterial occlusion persisting for about twenty-four hours causes gangrene, occlusion for shorter periods, or less complete occlusion, causes ischæmic contracture It is known that any source of arterial occlusion may be responsible, at any level, in any limb An unduly tight plaster may certainly be the cause, because a tight plaster may obstruct the arterial flow; but this is far from being the usual cause. In most cases ischæmic contracture is attributable to contusion of an artery, traumatic arterial spasm, thrombosis, embolism, perforation, rupture, traumatic aneurism, arteriovenous fistula, or any arterial injury or sequel of injury that reduces the blood supply of the muscles of a limb

Most recorded cases have been in the upper limb and they have involved the brachial artery at the level of a supracondylar fracture of the humerus Backward displacement of the small fragment stretches the artery across the fracture-site causing contusion, laceration or complete severance In 30 per cent. of recorded cases the radial or ulnar arteries have been injured by fractures of the shafts of one or both forearm bones Other fractures of the upper limb have been responsible in a few cases Figure 212 is the arteriogram of a fracture of the neck of the humerus with ischæmic contracture from injury of the axillary artery. In a fracture of the clavicle which was treated in 1930 a short inner fragment was forced directly backwards and lay at right angles to the skin occluding the subclavian artery, the limb was pulseless and cold, and although the circulation was improved by replacement of the clavicular fragment, ischæmic contracture supervened. Fractures near the wrist may damage the radial artery and cause reflex vasospasm of the whole length of the vessel throughout the forearm, and even of the brachial artery to the axilla, thus causing ischæmic contracture

[1] Brooks, B *Arch Surg*, 1922, 5, 188
[2] Jepson, P N *Ann Surg*, 1926, 84, 785
[3] Middleton, D S *Brit J Surg*, 1930, 18, 188
[4] Volkmann, R von *Zbl Chir*, 1881, 8, 801 (reprinted, *J int Chir*, 1938, 3, 77)

of muscles proximal to the level of arterial injury I have seen two patients with ischæmic contracture of the forearm developing after osteotomies of the lower end of the radius for mal-united Colles' fracture. In the lower limb, cases have been reported after fracture of the upper shaft of the tibia, fracture of the neck of the fibula, traumatic rupture of the popliteal artery, perforation of the popliteal artery and embolism of the femoral artery [1]



Fig. 212

Fracture neck of humerus with ischæmic contracture

An arteriogram shows occlusion of the axillary artery at the level where it was struck by the lower fragment of the humerus at the time of displacement of the fracture The ischæmia caused Volkmann's contracture

A child, aged seven years, with bilateral compound fractures of the shafts of the femora treated in an unpadded double-plaster spica which was applied within one hour of injury, developed bilateral ischæmic contracture of the lower limbs, the complication in this case being due to the tight plaster

Morbid anatomy : *Muscles*—Loss of the blood supply of a muscle causes necrosis and a "muscle sequestrum" [2,3] Exactly as in the case of a bone

[1] Jefferson, G *Brit. med J*, 1934, 2, 1090 Griffiths, D *Lancet*, 1938, 2, 1339
[2] Bristow, W R *Brit J Surg*, 1923, 10, 475
[3] Griffiths, D "Volkmann's Ischæmic Contracture" *Brit J Surg*, 1940, 28, 244

sequestrum, microscopic examination shows that the general contour is preserved, but the tissue is dead; individual muscle fibres can be recognised but there are no living nuclei either in muscle fibres or in interfibrillary tissue (Fig 214) Interfibrillary fibrosis, such as occurs after denervation or infection of a muscle, is never seen in ischæmic contracture; the tissue is inactive; it is dead Surrounding the necrotic area there is phagocytic and fibroblastic cellular activity In the course of time, fibrosis proceeds from without inwards and replaces the dead muscle with fibrous tissue which contracts and shortens *Nerves*—The same injury which bruises or tears the brachial artery may also bruise or tear the median, ulnar or musculo-spiral nerves Even when there is no gross damage to nerve trunks, the limb may be paralysed by ischæmia of the nerve endings, and in severe cases



Fig 213

Normal muscle

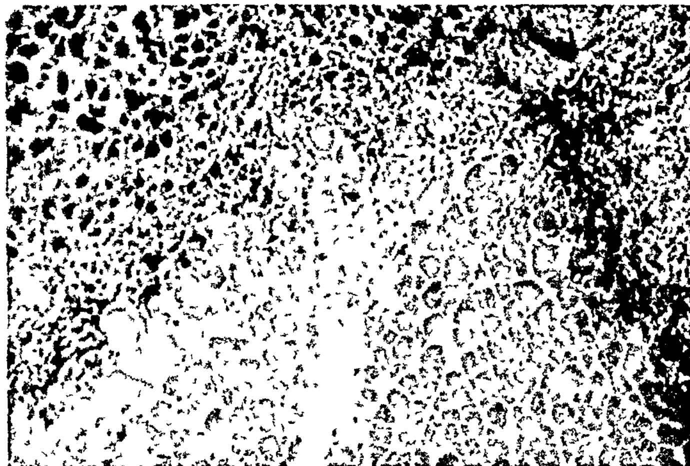


Fig 214

Muscle sequestrum in ischæmic contracture

Section of muscle after ischæmic contracture showing a typical "muscle sequestrum" The muscle fibres show normal contour, but they are dead; there are no nuclei There is fibroblastic cellular activity surrounding the sequestrum, but no interfibrillary fibrosis such as occurs in denervated or infected muscle.

by actual degeneration of the nerve trunks due to ischæmia [1] Many cases of Volkmann's contracture are therefore associated with nerve paralysis. When the nerve lesion is due to ischæmia alone it usually recovers spontaneously, first in the posterior interosseous nerve and later in the median and ulnar nerves

Clinical features of established contracture (upper limb)—Shortening of the forearm muscles causes typical deformity, with extension or hyperextension contracture of the metacarpo-phalangeal joints and flexion contracture of the interphalangeal joints [2] (Fig 215) In mild cases the contracture may be localised to one or two fingers. The flexion contracture of the fingers is increased when the wrist is dorsiflexed and reduced when the wrist is flexed (thus distinguishing it from flexion deformity due to capsular injury in the finger itself) This is the basis of the technique of correction described by Robert Jones With the wrist flexed, straight

[1] Leveuf, J "Contusion de l'Artère Humerale" *J Chir*, 1938, 2, 177

[2] It is to be recognised that the long flexor muscles of the forearm flex the interphalangeal joints, but have little power over the metacarpo-phalangeal joints which are flexed by the intrinsic muscles of the hand The extensor muscles of the forearm extend the metacarpo-phalangeal joints, but have little power over the interphalangeal joints which are extended by the interossei It follows that if long flexors and extensors of the forearm are simultaneously shortened, the fingers remain extended at the metacarpo-phalangeal joints but flexed at the interphalangeal joints

spatula splints are applied to each finger; a full-length splint is then applied to the forearm and hand and the wrist is gradually dorsiflexed. A still better method of correction is to support the wrist in a splint in a very slightly

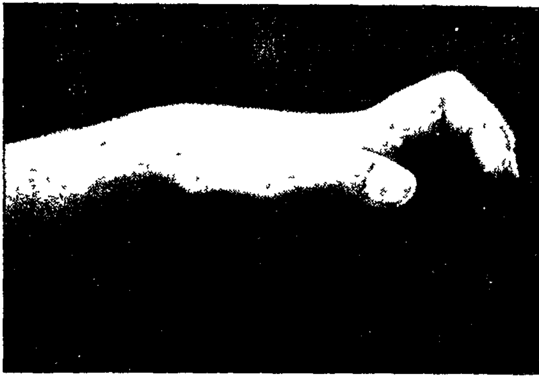




Fig. 215

Fig. 216

Typical deformity in Volkmann's ischæmic contracture (due in this case to brachial artery contusion in a supracondylar fracture of the humerus)

dorsiflexed position, and apply elastic traction loops to each individual finger. Treatment must be continued for a long time and improvement in the degree of contracture may be expected for at least two years Residual deformity may then be treated by lengthening the tendons in the lower forearm or by shortening the forearm bones A muscle slide of the forearm

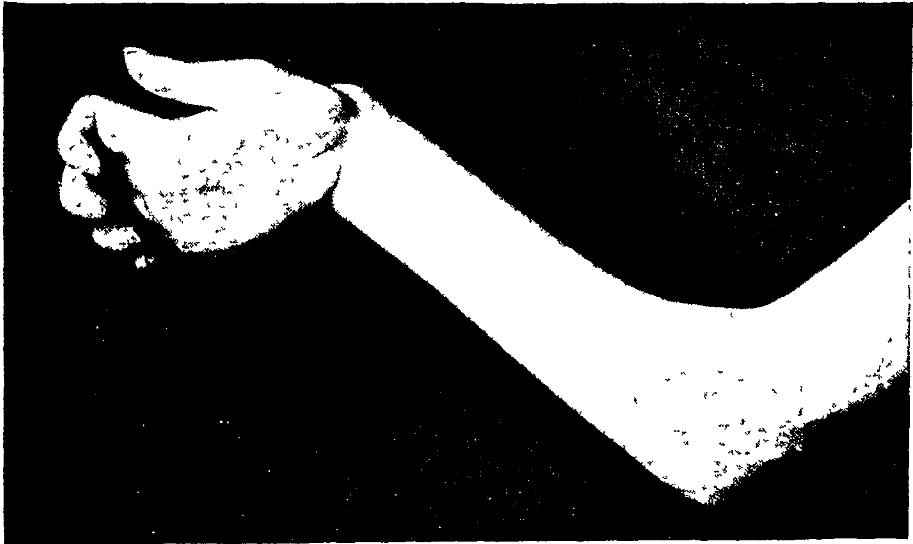


Fig 217

A severe case of Volkmann's ischæmic contracture with posterior interosseous, median and ulnar paralysis and permanent crippling despite many operations and years of treatment

muscles from the medial epicondyle of the humerus was advocated many years ago by Max Page,[1] with the object of correcting shortening of the flexor muscles, but the results were disappointing Recently a much more radical muscle slide has been claimed to be successful by Scaglietti [2] Despite all these measures severe Volkmann's contracture causes permanent crippling.

[1] Page C M *J Bone Joint Surg*, 1923, 5, 233
[2] Scaglietti, O *J Bone Joint Surg*, 1948, 30-B, 729

Main-en-griffe and ape-thumb deformities due to ulnar and median paralysis may be superimposed on flexion contracture due to fibrosis of the forearm muscles. Figure 217 represents the result of many operations and years of treatment in a particularly severe case, and it is the best possible illustration of the importance of preventive treatment

Early clinical diagnosis—The diagnosis should be made within the first hour The characteristic symptoms and signs may be summarised in the mnemonic list—pain, pallor, paralysis and pulselessness [1] Pain is variable, pallor is the first change but there may also be cyanosis, paralysis is of relatively late onset, but pulselessness—loss of the distal radial or tibial pulse—is immediate and invariable In the upper limb, the one clinical sign of importance is absence of the radial pulse The fingers are usually cold, slightly swollen and either cyanosed or pale There is sometimes burning or tingling pain in the forearm and hand, but the onset is often painless Insensitivity of the fingers and immobility in a slightly flexed position are later signs If there is actual flexion contracture the diagnosis has been made too late, the damage has been done

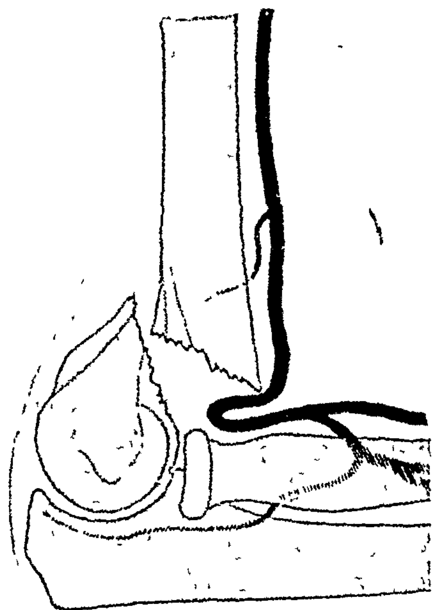


FIG 218

Flexion of a swollen elbow compresses the brachial artery, especially if there is an unreduced supracondylar fracture

Preventive treatment—Absence of the radial pulse in fractures of the elbow or forearm must be regarded with grave suspicion. If there is the slightest sign of circulatory embarrassment, active treatment must begin at once—it is urgent

1 Anæsthetise the brachial plexus with 2 per cent novocaine Not only is this a suitable anæsthetic for subsequent procedures, but it is a therapeutic measure that may avert the catastrophe

2 If there is a fracture of the elbow joint still unreduced, reduce it at once by manipulation Reduction of the fracture often relieves the vascular complication (Fig 218)

3. Exclude the possibility of external pressure by cutting through the whole length of any plaster, strapping or bandage encircling the limb. In cutting the bandage make certain that skin is exposed, wool beneath the bandage, soaked in clotted blood, may be more harmful than the bandage itself

4 If the elbow is swollen, and has been immobilised in flexion, support it in a less degree of flexion in order to prevent pressure in the antecubital space If necessary extend the elbow beyond the right angle and maintain immobility and reduction of the fracture by means of a posterior plaster slab

5 If despite these precautions the pulse remains absent and circulation is obviously impaired, do not wait more than an hour or two Expose

[1] Griffiths, D Ll *J Bone Joint Surg*, 1948, 30-B, 280

the artery at the level of injury Carefully avoid damage to all branches Raise the vessel from its bed, clean it of areolar tissue and apply warm saline douches Gently massage the vessel If the pulse does not return, inject papaverine (¼ gr) intravenously If the vessel is actually torn or ruptured apply ligatures above and below the lesion and resect not less than half an inch of the injured segment Do not, however, resect the vessel unless there is definite rupture Arteriectomy for simple spasm is of doubtful value

Ischæmic contracture in the lower limb—Injury to the popliteal and femoral arteries usually causes gangrene rather than ischæmic contracture Nevertheless ischæmic contracture does occur in the lower limb and, in gunshot wounds and infected fractures which have been treated in tight plaster, this complication is more common than is generally recognised. Arterial injury is not the only cause of ischæmia, tight plasters cannot be exonerated In recent years I have seen many infected compound fractures of the tibia which had been treated from an early stage in plasters fitting so closely that circulation of the toes was impaired. There is often loss of active movement of the toes even when no nerve has been injured; the toes become flexed and contracted, the deformity is resistant, and despite good union of the fracture, incapacity is prolonged This complication is due to obstruction of the arterial flow by a tight unpadded plaster causing ischæmic contracture of the muscles.

The complication is to be avoided by greater care in the use of the unpadded plaster cast, and by the more general use of elevation of the limb for several days or weeks after injury in order that the swelling shall be controlled An unpadded plaster should not be applied immediately after injury, after operative reduction of a fracture, after sequestrectomy, or after any other treatment of an infected wound that will be followed by reaction. In all these cases a wool bandage should be applied smoothly and the use of a non-padded cast should be deferred for several weeks The circulation of the toes and the power of active mobility must be watched with care. At the first sign of pallor, cyanosis, or immobility, the plaster must be cut throughout its length, if necessary it must be bivalved.

Ischæmic contracture of the lower limb has also occurred in simple fractures of the shaft of the femur treated in a Thomas' splint The pressure of rigid calico bandage encircling the limb to support the adhesive traction tapes was almost certainly the cause of the vascular obstruction.

GANGRENE FROM FRACTURES

Unpadded plaster casts—The application of an unpadded plaster cast to a fractured limb within a short time of injury, and before reactionary swelling has occurred, is dangerous Pressure within the rigid cast may become so great as to obstruct the arterial flow and cause not only ischæmic contracture but even gangrene. At this early stage, plasters should be padded with wool bandage, the unpadded cast being applied only after several weeks. Whether the plaster is padded or unpadded, the circulation of fingers and toes must be watched carefully and tested frequently It is not enough to apply digital pressure and confirm that the anæmic area refills with blood when pressure is withdrawn, this may be observed even after

complete circulatory stasis, because some blood remains in the digit The surgeon must be satisfied that the return of blood is brisk, that the digit is warm, that it is pink, and that it is neither cyanosed nor pallid. Unless he is satisfied the plaster must be bivalved *at once* and the front half removed. If the circulation does not return, and the pulse cannot be felt, the diagnosis of arterial injury must be accepted and the necessary steps be taken.





FIGS. 219-220

Bilateral gangrene of the lower limbs from combined fractures and third-degree burns of the limbs, treated in plaster casts

Gangrene from fractures and burns—The more severe the injury, and the greater the source of œdema, the greater is the peril of an unpadded plaster The peak of danger is reached when a major fracture is associated with a third-degree burn—an association that is not infrequent after aircraft crash-landings (Figs 219-220). To the hæmorrhage and swelling of the

fracture is added the reactionary œdema of the burn, and swelling is of such a degree that the application of a plaster cast, whether padded or unpadded, spells almost certain death to the limb. There were many such combined injuries in the Royal Air Force orthopædic service during the recent war, and I cannot recall one case treated in plaster that escaped amputation. Moreover there is a second danger, no less great than that of reactionary swelling, namely lymphatic spread of infection from the burn to the undrained hæmatoma of the fracture. It is clear that primary treatment must not be dominated by the problem of bone injury. The first objective must be to save the limb, to control infection, and to replace the skin. After that there is ample time to consider bone reconstruction and the correction of deformity.

Contusion of arteries by fracture—The types of arterial injury that may result from impact of the fragments of a fractured bone have already been discussed: they include concussion of the artery with traumatic spasm; contusion with spasm; contusion with thrombosis and spasm; embolism with spasm; and perforation or rupture with hæmatoma, aneurism or arteriovenous fistula. Of these injuries the most common is simple contusion with reflex vasospasm. In the upper limb the radial and ulnar arteries may be contused by the forearm bones, the brachial artery by the lower end of the humerus, the axillary artery by the neck of the humerus, or the subclavian artery by the clavicle. The potential collateral circulation in the upper limb is good and, although reflex vasospasm may cause ischæmic contracture, it is seldom that there is complete and persistent arrest of the circulation causing gangrene. In the lower limb the risk of gangrene is twice as great.[1] The femoral artery is in danger where it is anchored close to the femur by the fibrous arch of the adductor magnus, and still greater is the danger to which the popliteal artery is exposed in the lower part of the popliteal space.

Gangrene after high oblique fracture of the tibia and fibula—The popliteal artery, at its bifurcation, lies close to the tibia and in actual contact with the fibula, the neck of this bone being grooved by the anterior tibial branch. The vessel is anchored by the fibrous arch of the soleus and by the passage of the anterior tibial branch over the interosseus membrane. If there is a fracture of adjacent bones the vessel cannot easily escape contusion or traction injury. The bone injury which is nearly always responsible is a high fracture of both leg bones, oblique from before, upwards and backwards; the main shaft fragment is displaced upwards and backwards and the artery is impaled upon its sharp margin (Fig. 221). Of seven fractures of this type and with this displacement that I have seen, no less than five were complicated by gangrene of the foot and leg[2] (Fig. 222). This frequency is the more striking when it is recalled that after searching the literature Dodd could find only one case in twenty years (1914-34). It is probable that cases were not then recognised or reported, but the greater use of the unpadded plaster cast in recent years cannot be ignored. The collateral

[1] Dodd, H. "Gangrene following Fractures." *Brit. J. Surg.*, 1934, 22, 256.

[2] One other case has a direct bearing. I operated on a nurse aged 28 for severe genu varum due to arrested growth in adolescence of the inner part of the tibial epiphysis. The bone was almost but not quite divided with an osteotome; the fracture was completed manually, the deformity corrected and a padded plaster applied. The circulation of the toes was believed by the resident surgical officer to be satisfactory until the third day when they were obviously pulseless. Exploration of the popliteal artery showed no bruising or laceration, but it was strongly contracted at the level of the fibrous arch of the soleus. Extensive mobilisation and stripping did not restore the circulation, and three days later a gangrenous limb was amputated. Examination of the amputated limb confirmed that the arterial lesion had been a simple traumatic vasospasm with no gross injury or thrombosis.

circulation after injury to the popliteal artery, at a level below the anastomoses of the knee, depends upon small and insignificant vessels. The pressure of plaster, even if carefully applied, may well hinder the development of so feeble a circulation

High fracture of the shafts of tibia and fibula should therefore be recognised as a dangerous injury The possibility of arterial damage must be anticipated and the application of plaster be withheld for several days The position of



Fig 221

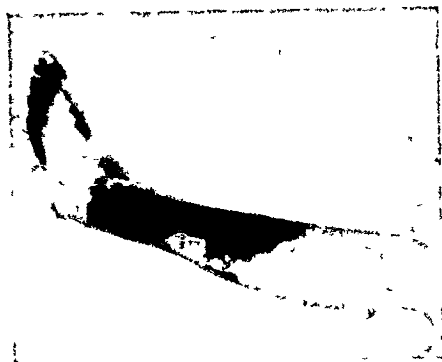


Fig 222

Gangrene from high oblique fracture of the tibia

From the point of view of vascular complication, this is one of the most dangerous of all fractures Five out of seven fractures of this type were complicated by gangrene The main fragment of the tibia is displaced upwards and backwards, and its sharp upper margin strikes the popliteal artery at its bifurcation, a level where it is anchored and cannot escape Anticipate the danger. Defer the application of plaster for three days If the tibial pulse cannot be felt, block the sympathetic with novocaine. Prepare to expose the artery

the fragments can be controlled, and redisplacement prevented, by skeletal traction from a pin in the lower shaft of the tibia, the limb being supported in a Thomas' splint The tibial pulse and the circulation of the toes must be watched with great care At the first sign of circulatory failure a spinal anæsthetic should be given (or a paravertebral novocaine injection) and the popliteal artery exposed at its bifurcation by splitting the upper fibres of the soleus If stripping the vessel, removing the areolar tissue, and injecting papaverine do not restore the circulation, arteriectomy must be considered, particularly when there is actual bruising or thrombosis of the vessel

In the treatment of other injuries of the leg, the surgeon should remember the susceptibility of the popliteal artery and its branches Contusion of the anterior tibial artery against the neck of the fibula has caused gangrene from "bumper injuries" even when there was no fracture It is obvious, therefore,



Fig 223

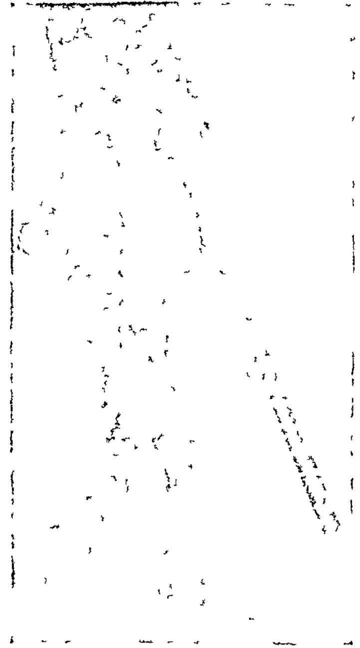


Fig 224

Fracture of the lateral tuberosity of the tibia from a motor-cycle accident The fragment was tilted backwards and it perforated the popliteal artery. The limb was amputated through the lower third of the thigh on the fifth day after injury The specimen shows perforation of the artery (Fig 224) *(Treated in the Orthopædic Service of the Royal Air Force by Squadron-Leader Small and Squadron-Leader Shields)*



Fig 225

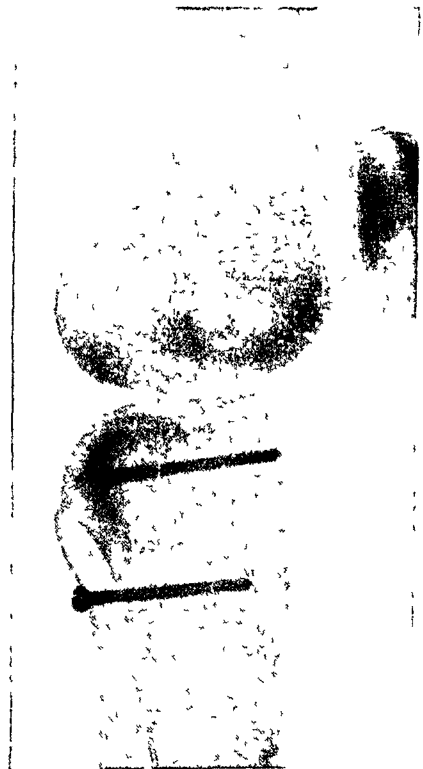


Fig 226

Similar fracture of the lateral tuberosity of the tibia with backward tilting of the fragment in which the circulation was imperilled without the artery being perforated. Open reduction was performed by Mr Crawford Adams, the fragment of the tuberosity being fixed with one screw and the tibio-fibular diastasis with another Vascular damage was avoided *(Treated in the Orthopædic Service of the Royal Air Force)*

that the technique of reducing fractures of the tibial tuberosity by tightly encircling the fragments with a tourniquet and hammering them with a sandbag is to be regarded with grave suspicion One case has been recorded where this treatment was complicated by gangrene Fractures of the tibial tuberosity in which the fragment is displaced backwards must be regarded with suspicion (Figs 223-226) There must also be care in the reduction of tibial shaft fractures by traction apparatus, where counterpressure is applied in the popliteal space.

GANGRENE FROM THE USE OF TOURNIQUETS

More limbs have been lost by the use of tourniquets than have been saved. The correct first-aid treatment of hæmorrhage from a wound is often to



Fig 227

Gangrene due to a tourniquet

A wrist operation was performed with a flat Esmarch rubber tourniquet on the upper arm, it was removed in forty-five minutes Persistent traumatic arterial spasm caused gangrene Beware tourniquets! Use only the pneumatic type

keep the patient at rest and do no more Sometimes it is necessary to maintain pressure over a large pad applied to the wound itself In teaching first-aid and ambulance workers, this treatment is usually dismissed in a few words, hours are then devoted to the consideration of "pressure points" and the application of tourniquets It is to be expected, therefore, and it is in fact the case, that when an ambulance man sees a wound he at once thinks of a tourniquet If a tourniquet is not applied tightly enough, the veins are obstructed but not the arteries I have vivid recollection of a child who for this reason almost bled to death, hæmorrhage from the wound ceased as soon as the tourniquet was released, the tourniquet had been far more perilous than the wound On the other hand, if the unfortunate ambulance worker applies the tourniquet too tightly he is more than likely to cause gangrene It is not enough to mark the patient's forehead with a T and remove the tourniquet at intervals, because if traumatic arterial spasm is once induced it continues whether the tourniquet is removed or not. Vasospasm, ischæmic contracture and gangrene from the application of

tourniquets have now been recorded by Esmarch,[1] Wallis,[2] Ducastaing,[3] Griffiths,[4] Trueta and Barnes,[5] and Cohen.[6] The danger arises not from the length of time that the tourniquet is in position but from the force with which it is applied. Gangrene necessitating amputation of the arm has arisen from the use of an Esmarch rubber tourniquet for no more than forty-five minutes (Fig. 227). There is certainly greater danger in the upper limb than in the lower limb, but in neither upper nor lower limb is there complete safety except with the controlled pressure of a pneumatic tourniquet. The partly skilled first-aid worker who has been taught to improvise tourniquets from bandage or cord, tightened with pieces of wood, is a most dangerous person. There is danger in applying the tourniquet too tightly; there is danger in not applying it tightly enough; there is danger of killing the patient by the renal complications of tourniquet pressure; the only danger that is remote is that the patient will bleed to death for lack of a tourniquet. It has been known for two hundred years that completely severed arteries cease bleeding within a few minutes. First-aid workers should use local pressure on the wound. Surgeons should use pneumatic tourniquets.

Abolish tourniquets from first-aid equipment!

IMMERSION FOOT, SHELTER FOOT, TRENCH FOOT

A pilot crashed in the North Sea. For fourteen days and fourteen nights he floated in a rubber dinghy, awash with the waves, cold, wet, benumbed. He had no food, and on the eighth day he drank his last ration of water. He could not have survived but for an incredible chance. On the twelfth morning a weary sea-gull, seeking refuge, hovered and landed on the edge of the perilous craft. The airman showed restraint; he waited, and when the bird raised its wing he clutched and caught it. With relish and no aversion he ate the bird; he sucked its brains and enjoyed its blood; he ate a small fish in its belly. On the fourteenth night in the moonlight he saw a motor torpedo boat. He stood on his feet, waved and shouted, and was taken aboard in great spirits. He stood on his feet—but they were gangrenous (Fig. 228). Five hundred hours of continuous exposure to cold and wet had caused immersion feet,[7,8] a peril to which airmen are exposed in dinghies, sailors in life-boats, soldiers in trenches and bombed civilians in dug-outs.[9,10] The circulatory failure is allied to frost-bite and is due to arterial spasm induced by cold. Contributory factors are damp, venous congestion or thrombosis which aggravate the arterial spasm, and exposure, hunger or debility which lower the general circulation. The feet become cyanosed and blue. In mild cases the circulation may recover, but in more severe cases gangrene develops in the toes and over the heels and malleoli. The feet at first look worse than they are, and if conservative treatment is adopted it will often be found that the gangrene is only skin-deep and that

[1] Esmarch, F. *C. R. Cong. méd. internat.*, 1877, 5, 308; *Brit. J. Surg.*, 1942, 30, 75.
[2] Wallis, F. P. *Practitioner*, 1901, 67, 429.
[3] Ducastaing, R. *Bull. Mem. Soc. Chir., Paris*, 1919, 45, 604.
[4] Griffiths, D. *Brit. J. Surg.*, 1940, 28, 256.
[5] Trueta, J., and Barnes, J. M. *Brit. J. Surg.*, 1942, 30, 74.
[6] Cohen, S. M. *Guy's Hosp. Rep.*, 1940, 90, 208.
[7] Ungley, C. C., and Blackwood, W. "Immersion Foot and Immersion Hand." *Lancet*, 1942, 2, 447.
[8] Webster, D. R., Woolhouse, F. M., and Johnston, J. L. "Immersion Foot." *J. Bone Joint Surg.*, 1942, 24, 785.
[9] Lake, N. C. "Effects of Cold upon the Body." *Lancet*, 1917, 2, 557.
[10] Knight, B. W. "Trenchfoot in Civilians." *Brit. med. J.*, 1940, 2, 610.

deeper structures are still alive In severe cases persistent vasospasm may cause thrombosis of the tibial arteries. In these patients, even if gangrene has been avoided, acutely painful vasoconstrictive attacks may persist over a period of months or years Leriche reported such crises in trench



Fig. 228

Immersion feet

Exposure to cold causes reflex vasospasm and sometimes thrombosis of the vessels, with gangrene of superficial tissue and toes This patient was a pilot, adrift in a dinghy for fourteen days. He was without water for six days His life was saved by a sea-gull

foot in patients who gained complete relief from pain and improvement in the circulation after resection of the thrombosed vessels [1]

FROST-BITE

Whereas immersion foot is due to vasospasm, frost-bite is due to actual freezing with the formation of minute crystals of ice in the skin and deeper tissues The effects on the tissues are still more destructive. During the recent war an American "Flying Fortress" was at a height of 8,000 feet where the temperature was many degrees below zero The bomb-aimer lay prone in the nose of the machine A chance fragment of anti-aircraft shell shattered the perspex, and in the fraction of a minute that it took the bomb-aimer to extricate himself, and escape from the blast of frozen air blowing in at many hundreds of miles an hour, his face was destroyed by frost-bite (Fig. 229) Similarly a member of the crew of a British "Wellington" removed his gloves in an attempt to meet the situation when a hole was blown in the side of the aircraft. Again there

[1] Leriche, R "The Surgery of Pain," translated by Archibald Young London Baillière, Tindall & Cox, 1939, 239

was an icy blast at many hundreds of miles an hour—and again there was complete destruction of tissue by frost-bite (Figs. 230-231).



FIG 229
Frost-bite of the face in the bomb-aimer of a "Flying Fortress"



FIG. 230
Frost-bite of the hands in the member of an air-crew



FIG 231
There was similar change in the right hand

CRUSH SYNDROME—"TRAUMATIC ŒDEMA"

The first accounts in English literature of the crush syndrome were published in 1941 at a time of intensive bombing, when many victims were buried for hours or days under fallen masonry and debris The same condition was recorded after the Messina earthquake as "acute pressure necrosis" The clinical picture is exactly similar to that seen after prolonged tourniquet pressure

Clinical features—As a rule there is no fracture, external wound or hæmorrhage, but only abrasion of the skin and discomfort on using the

muscles that have been crushed After a latent period, which may last for several hours, the blood pressure falls to severe shock levels. This is due to plasma loss into the damaged area, and it is accompanied by hæmoconcentration up to 160 per cent hæmoglobin, and œdema of the limb. Sometimes the circulation becomes impaired Arterial pulsation is diminished or even lost and there are signs of incipient gangrene

Renal changes—The urinary output is low from the beginning After a few days the output is progressively diminished and there are signs of renal failure. There is acidosis and myohæmoglobinuria The blood urea rises to 300 or 400 mg per cent, and death from uræmia occurs in 60 per cent of cases Post-mortem examination shows that the renal failure is due to necrosis of the distal convoluted tubules The appearances are similar to those seen in the kidneys of patients dying from mismatched blood transfusions where a hæmoglobin pigment, rendered insoluble by high concentration and an acid reaction, is precipitated in the tubules

Pathology—Studies in the pathology of the crush syndrome in Oxford by Trueta, Barclay, Franklin, Daniel and Prichard[1] were based on the recognition that prolonged application of a tourniquet to the thigh of a rabbit caused arterial spasm extending well above the level of arterial compression and even involving the main artery of the contralateral limb. It was thought that this vasospasm might even extend high enough to involve the renal vessels and thus explain the uræmia of the crush syndrome, and the death from uræmia of patients in whom a tourniquet had been left inadvertently on the lower limb for many hours Subsequent studies showed that there was more than renal vasoconstriction with diminution of blood flow—there was an actual short circuit of blood within the kidney. Trueta postulated that there are two potential circulations in the kidney and that, in response to sympathetic stimulation from injuries of the lower limb, blood is bypassed through the lesser of the two circulations, entirely escaping the glomeruli of the cortex and reaching the venous system without traversing the usual capillary network.

Such circulatory bypasses and arteriovenous shunts are not however peculiar to the kidney. Microscopic study of the circulation in living tissue shows that in the smaller vessels the immediate local response to injury is vascular stasis with packing of the red cells into dark homogeneous masses of clumped or agglutinated corpuscles which block many vessels, cause reversed flow in some, and direct arteriovenous shunt in others.[2-4] The local stasis is gradually resolved when columns of agglutinated cells are washed away and break off into the general circulation, thus causing embolic blockage of far-distant capillaries in the mesentery, conjunctiva and, indeed, every tissue that has been examined

Thus the renal complications of crush and tourniquet injury of the limbs may be the consequence not of any "dual circulation" of the kidney under sympathetic control but of simple embolic blocking of vessels of the renal cortex by agglutinated clumps of red cells liberated from the site of injury

[1] Trueta, J, Barclay, A E, Franklin, K J, Daniel, P M, and Prichard, M M L "Studies of the Renal Circulation" Oxford Blackwell Scientific Publications Ltd, 1947
[2] Chambers, R, Zweifach, B W, and Lowenstein, B E "The Peripheral Circulation during the Tourniquet Shock Syndrome in the Rat" *Ann Surg*, 1944, 120, 791
[3] Knisely, M H, Eliot, T S, and Bloch, E H "Sludged Blood in Traumatic Shock Microscopic Observations of the Precipitation and Agglutination of Blood Flowing through the Vessels in Crushed Tissues" *Arch Surg*, 1945, 51, 220
[4] Bigelow, W G, Heimbecker, R O, and Harrison, R C "Intravascular Agglutination (Sludged Blood), Vascular Stasis, and Sedimentation Rate of the Blood in Trauma" *Arch Surg*, 1949, 59, 667

CHAPTER VII

NERVE INJURIES

Peripheral nerve injuries were formerly classified into two groups: anatomical lesions in which the nerve was completely divided: and "physiological lesions" in which, despite an appearance of anatomical continuity, there was loss of conduction of the nerve. This subdivision was not entirely satisfactory. An anatomical lesion was clearly recognisable and was always associated with Wallerian degeneration in the distal nerve stump, but "physiological" lesions included different types of injury, some with Wallerian degeneration and some without. It is better therefore, to define three groups of nerve injury: complete divisions of the nerve; nerve lesions in continuity, and transient nerve blocks. Complete division of a nerve causes a break not only in the axons but also in the supporting structures epineurium, perineurium and endoneurium, and it is the lack of continuity of these structures that explains the difficulty of spontaneous regeneration. A lesion in continuity from severe compression or crushing of a nerve also interrupts the axons and causes Wallerian degeneration, but the supporting structures remain in continuity so that spontaneous recovery is the rule and regeneration occurs with greater speed and accuracy than after complete division. A transient nerve block from contusion or "concussion" of a nerve does not cause Wallerian degeneration; spontaneous recovery occurs more quickly than in any other type of lesion, and normal function is restored within a week or two. These three types of nerve injury may be described in the terms of a recent nomenclature as neurotmesis, axonotmesis and neurapraxia.[1]

TYPES OF NERVE INJURY

Complete nerve division—neurotmesis—Histological examination of the nerve trunk distal to a complete nerve lesion shows degeneration of axis cylinders, fragmentation and absorption of myelin sheaths, and proliferation of the neurolemmal cells of Schwann. The Schwann cells form columns which act as guiding tubes for the regenerating axons. At the cut surface of the nerve they grow out from the distal segment, apparently seeking to reach the proximal end of the nerve and direct the sprouting axons across the gap.[2] The nerve proximal to the lesion shows retrograde degeneration for about one centimetre, but regeneration begins within a few days with a growth from each divided axon of twenty or more streams of axoplasm. In their flow they separate from each other and join once more in accordance with the mechanical obstructions they may meet. If a completely severed nerve has not been sutured with the segments in accurate apposition, the

[1] "Tmesis"—cutting or division, as in tenotomy; hence neurotmesis implies division of the whole nerve, axonotmesis implies division of axons but not of the supporting structures of the nerve. "Apraxia"—non-action, hence neurapraxia implies transient loss of action or of conduction. These terms were suggested by Professor Henry Cohen and introduced by Professor H. J. Seddon, "A Classification of Nerve Injuries," *Brit. med. J.*, 1942, 2, 237.
[2] Young, J. Z., "Nerve Regeneration—Importance of the Peripheral Stump," *Lancet*, 1940, 2, 128.

intervening blood clot is transformed rapidly to scar tissue which offers an obstruction to the streams of axoplasm. They then wander aimlessly Some may penetrate the scar and reach the Schwann cell tubes of the distal segment, thus permitting limited recovery of function, others travel laterally, a few double back and twist round the central fibres This great axonal activity, together with cell proliferation at the end of the central stump, accounts for the well-known globular swelling which is a neuroma Similar cellular activity at the proximal end of the distal segment gives rise to a smaller glioma On the other hand, if accurate contact is established between the proximal and distal nerve segments, some of the streams from each axon flow into Schwann cell tubes Other streams from the same axon may wander abortively between the tubes but they gradually disappear, and the axonal sprout that was successful in hitting the target develops at the expense of its fellows. Thus there is a progressive reduction in the number of streams of axoplasm growing from one axon

The functional result is determined by the proportion of nerve fibres that regenerate successfully When apposition is inaccurate, many central axons fail to find a peripheral tube down which to grow, spontaneous regeneration is therefore unusual after complete division If the nerve ends are sutured in accurate apposition much better function is regained, provided that care is taken to avoid deep insertion of sutures, which would deflect the growth of axons into irregular whorls. Even when apposition is accurate, and there is no obstacle at the suture line, considerable criss-crossing of fibres is inevitable Central axons grow blindly down any available peripheral tube and, if the nerve trunk is mixed, motor axons may grow down sensory channels and sensory axons down motor channels The functional result is therefore imperfect, particularly, for example, after division of the median and sciatic nerves Regeneration in a simple nerve which includes only sensory fibres shows similar crossing in the inaccuracies of localisation which may remain permanently. In the musculo-spiral nerve, consisting largely of motor fibres, the result is relatively good. Regeneration occurs at a rate variously estimated from 1 to 4 mm a day. It is possible to make a reasonably accurate estimate of the time of expected recovery by measuring the length of nerve involved, allowing an average rate of growth of 2 mm a day, and adding ten days for crossing the suture line

Lesion in continuity—axonotmesis—If a nerve is compressed or crushed but not severed, and there is no loss of anatomical continuity, axis cylinders undergo Wallerian degeneration as they do after complete division, and proliferation of Schwann cells at the level of injury gives rise to a fusiform enlargement ("neuroma" or "neuroglioma") But the gap between proximal and distal segments of the nerve is no more than a break in the axons Regeneration occurs spontaneously and at a considerably faster rate than after complete division. Moreover, the general architecture of the nerve is preserved at the level of injury, and central axons grow into appropriate peripheral channels with minimal crossing of fibres, so that good functional recovery occurs even in mixed nerves.

Transient block—neurapraxia—Simple contusion, concussion or traction may cause temporary loss of conduction of a nerve, with no actual degeneration of axis cylinders in the distal segment. The lesion is often incomplete Subjective alteration of sensation such as tingling or

numbness may be the only disability. Sometimes there is complete motor loss with incomplete sensory loss. Recovery takes place within a week or two, and often occurs with equal rapidity throughout the whole area of distribution.

Treatment of nerve lesions—Paralysis due to transient nerve block recovers spontaneously and no special treatment is needed. Paralysis due to a lesion in continuity usually recovers spontaneously and exploration of the nerve is unnecessary Paralysis due to complete division seldom recovers spontaneously and suture of the nerve is essential. The differentiation between these lesions cannot be established in early days with any certainty, and exploration is sometimes advisable in order to determine the type of injury The probabilities can be summarised thus .

1. If there is an open wound and paralysis is complete, the nerve is probably divided and nerve suture is necessary.
2. If there is an open wound and paralysis is incomplete, the symptoms may be due to a lesion in continuity or transient block which will recover without operation, but they may also be due to complete division of part of the nerve, which necessitates suture Unless recovery is prompt, exploration is advisable
3. If there is a closed fracture of the type often associated with nerve concussion, contusion or traction (*e g*, ulnar paralysis in elbow injuries), the paralysis is probably due to transient block and operation is not necessary
4. If there is a closed fracture of the type often associated with severe nerve contusion or laceration (*e g*, musculospiral paralysis in fractures of the humerus, or median paralysis in supracondylar fractures), the nerve injury is probably a lesion in continuity, but it may be a complete division, and unless there are signs of recovery within three or four months, exploration is advisable

The best time for exploring a nerve is to be determined primarily by the importance of avoiding infection. The operation must not be performed until it is reasonably certain that the wound will heal by first intention. Within limits "the interval between the wound and the operation has no influence on the time taken for recovery"[1] There is certainly no harm in waiting six months, but it must be recognised that the longer operation is delayed the more serious are the changes in the paralysed limb[2] Moreover, if the interval is greater than eighteen months, the chances of successful recovery are slight[3]

NERVE INJURIES IN WOUNDS

Recent wounds—Divided nerves should seldom be sutured at the time of the original operation of wound excision unless it is almost certain that the wound will heal by first intention If there is the slightest risk of infection there can be no justification for nerve suture The primary operation should be concentrated on the task of minimising infection and securing the most rapid possible healing If the wound heals without infection the nerve can be explored within two or three weeks; otherwise nerve suture

[1] Medical Research Council Committee on Injuries of the Nervous System, 1920 Special Reports M R C, No 54

[2] Cairns, H, and Young, J Z "Gunshot Wounds of Peripheral Nerves" *Lancet*, 1940, 2, 123

[3] Platt, H "Surgery of Peripheral Nerve Injuries of Warfare" Bristol John Wright & Sons Ltd, 1921

should be deferred until about two months after healing is sound If the wound is associated with a fracture, suture of the nerve should usually be delayed until the fracture is united and neighbouring joints are mobilised

Exploration of nerve and neurolysis—A generous incision is made, and the nerve is identified above and below the injury before any attempt is made to trace it through scar tissue at the level of injury If the nerve is found to be divided completely, with a well-defined proximal neuroma and distal glioma, there can be only one decision[1], the nerve bulbs must be excised and the nerve sutured If, however, there is anatomical continuity and a fusiform enlargement, the decision must be made as to whether it is a lesion in continuity not calling for excision and suture, or a complete division in which proximal and distal bulbs are joined by scar tissue into a single fusiform spindle If the central part of the expansion is hard and dense the division is very probably complete. This may be confirmed by electrical stimulation when the lesion is complete, a proximal stimulus will fail to cause contraction of muscles innervated from a lower level, a distal stimulus will not give rise to any sensation of tingling in the area supplied by the nerve If the surgeon is in doubt, it is better to be satisfied with the neurolysis already completed than to make the error of excising a regenerating nerve The guiding principle should be "radical nerve exploration — conservative nerve operation"[2]

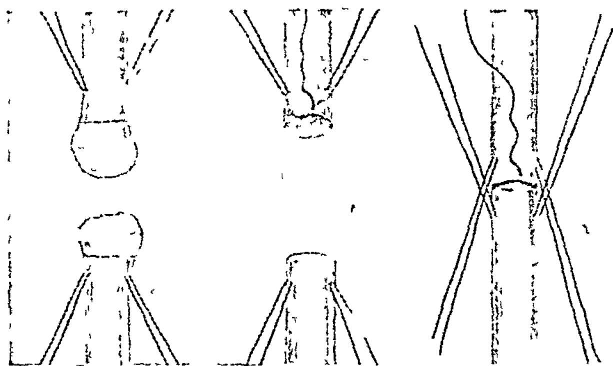


FIG 232

Suture of severed nerve The proximal neuroma and distal glioma are excised Guide sutures in the sheath may be used for orientation and approximation The finest thread or silk stitches are inserted through the nerve sheath The guide sutures are then removed

Suture of nerve—The nerve ends should be mobilised with such a degree of freedom that after the nerve bulbs have been excised there is easy contact without tension. It is usually necessary to expose a considerable length of nerve, to flex the joints above and below, and sometimes to gain length by transposing the nerve to a new bed (for example, transposing the ulnar nerve to the front of the elbow joint). The proximal neuroma and distal glioma must be cut back with a safety-razor blade or sharp scalpel until the whole area of intraneural scarring has been removed, the typical faggot-like leash of nerve fibres being exposed and the nerve sheath showing normal retractability The nerve ends are then correctly orientated and guide sutures are inserted by which to hold them in accurate apposition Fine linen thread or fine silk (No 0 Deknatel) are better suture materials than non-chromicised 000,000 catgut The sutures must include the nerve sheath only and not be placed so deeply as to disturb the underlying axis cylinders There must be no crowding or compression of the fibres and interrupted sutures are therefore

[1] "**Bulb suture**"—The exception to this observation—that there is one decision and one only—arises in a case where, despite full mobilisation of the nerve and flexion of neighbouring joints, the gap to be bridged will be too great for immediate suture without tension Bulb suture is then advisable, the nerve bulbs being brought together by full flexion of the joints and secured by a heavy stitch through the scar tissue Cautious and gradual stretching after this preliminary operation may give sufficient length to permit excision of the bulbs and formal suture at a later date

[2] Stookey, B *Neurol Bull*, 1919, 2, 380

better than a continuous stitch The fibres must just touch ; if th be any defect at all, it should be a slight gap rather than a crowdi fibre must be enclosed within the sheath so that it is in apposit opposite fibre The repaired nerve is then replaced in a muscl from scar tissue The flexed position of the joints, by which apposition o the nerve ends and freedom from tension was secured must be maintained during suture of the wound and for two or three weeks after operation by means of a light plaster cast.

Fibrin suture of nerves—"The least irritant sutures are those which are not there" (Sterling Bunnell) Suture material, no matter how fine or how carefully inserted, is a mechanical obstacle which tends to interrupt the line of growth of budding axons and deflect them from their course A method has been devised of holding nerve ends together with a "glue" of concentrated blood plasma, coagulated by the addition of chicken-embryo tissue-extract [1] Within about two minutes of adding the extract, the plasma clots to a jelly This holds the nerve ends in apposition and allows axons to bridge the gap with greater accuracy and speed than they can cross a scar sutured with thread or catgut It must be recognised, however, that this method of securing apposition of the segments of a divided nerve is not yet of proved reliability

NERVE INJURIES IN CLOSED FRACTURES AND DISLOCATIONS

Nerve injuries that complicate fractures and dislocations may be classified chronologically into three groups : (1) primary nerve injuries sustained at the time of bone injury by ischæmia, compression, contusion, traction or laceration of the nerve ; (2) secondary nerve injuries developing after some weeks or months as the result of friction or late compression ; (3) delayed nerve injuries developing after many years from stretching or compression of the nerve in consequence of deformity, bone thickening or osteophyte formation from late arthritis

1. FRACTURES WITH PRIMARY NERVE INJURIES

Ischæmic nerve lesions—These have already been considered under Volkmann's ischæmic contracture (p 113) Loss of blood supply for thirty minutes causes temporary paralysis of the sensory and motor nerve endings Ischæmia for a longer period may cause Wallerian degeneration, but it is a lesion in continuity which recovers spontaneously within a few months

Compression nerve lesions—Reference has been made to compression injuries of nerves accompanying crush œdema in air-raid victims whose limbs are crushed for many hours by fallen masonry Paralysis may occur even when the more serious crush syndrome is avoided The external popliteal, sciatic, musculospiral or ulnar nerves are usually involved, the nerve being compressed between the adjacent bone and the fallen weight The injury is a lesion in continuity and recovery is spontaneous

Contusion nerve lesions—A nerve may be contused by a dislocated or fractured bone When the carpal lunate bone is dislocated forwards it is forced into the confined space beneath the anterior annular ligament, and the median nerve is often injured by the blow [2] (Fig 233) The paralysis is

[1] Young, J Z, and Medawar, P B "Fibrin Suture of Peripheral Nerves" *Lancet*, 1940, 2, 126
[2] Watson-Jones, R "Carpal Semilunar Dislocation with Nerve Lesion" *Proc Roy Soc Med* (Section of Orthopædics), 1929, 22, 1071

incomplete and due to transient block, when injury is more severe it is a lesion ... y which recovers fully In about 15 per ... f supracondylar fractures the median or ulnar nerves are contused by the lower margin of the main fragment of the humerus[1] (Fig 234) As a rule recovery is complete within a few weeks neurapraxia) or at the most within about three months (axonotmesis), and no treatment is required other than reduction of the fracture Occasionally, however, the nerve is completely severed even in closed fractures



Fig 233

Dislocated lunate bone with median paralysis due to contusion of the nerve.

Traction nerve lesions—Traction nerve lesions causing transient nerve block are very common in fractures and dislocations, particularly of the elbow joint where the ulnar nerve is involved, and the shoulder joint where the brachial plexus is injured. More severe traction injury causing a lesion in continuity sometimes occurs in shoulder dislocations, and in injuries of the knee joint which stretch the lateral popliteal nerve Traction causing complete division of nerves seldom occurs except in supraclavicular injuries of brachial plexus

Ulnar paralysis—The ulnar nerve is held in the post-condylar groove by a roof of fibrous tissue and a thin "mesentery." It is fixed to the forearm by its muscular branches If the elbow joint is forced into cubitus valgus the nerve is stretched Valgus deformity of the elbow often occurs as the result of a fall on the outstretched hand which may cause avulsion of the epiphysis of the internal epicondyle, outward subluxation or dislocation of the elbow, or a supracondylar fracture with outward displacement of the lower fragment (Figs 235-239). In these injuries all the structures on the inner side of the joint are stretched and ulnar paralysis is frequent[2,3]



Fig 234

Supracondylar fracture of humerus with median paralysis due to nerve contusion.

Similarly, if the forearm bones are displaced forwards, the nerve is stretched over the back of the lower end of the humerus Supracondylar fractures with forward displacement, and forward dislocation of the elbow with fracture of the olecranon, are unusual injuries, but when they occur there is a high incidence of ulnar paralysis On the other hand, if

[1] Bristow, W R "Complications of Supracondylar Fracture" *Brit J Surg*, 1923, **10**, 475

[2] Platt, H "Peripheral Nerve Complications of Fractures and Dislocations of the Elbow" Robert Jones Birthday Volume, Oxford Univ Press, 1928, 195

[3] Watson-Jones, R "Primary Nerve Lesions in Injuries of the Elbow and Wrist" *J Bone Joint Surg* 1930, **12**, 121



Fig 235



Fig 236

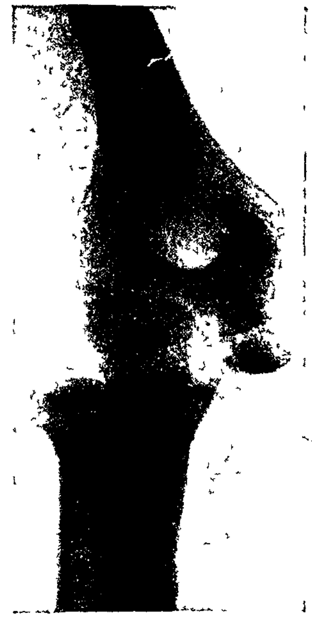


Fig. 237



Fig 238



Fig 239

Injuries of the elbow with ulnar paralysis due to traction injury

Fig 235—Avulsion of epiphysis of internal epicondyle Fig 236—Subluxation of elbow with inclusion of epicondyle on inner side Fig 237—Outward dislocation of elbow Fig 238—Supracondylar fracture with outward displacement Fig 239—Supracondylar fracture with forward displacement.

there is backward displacement of the forearm bones the nerve is displaced backwards from its groove, it gains free mobility, and escapes traction injury Ulnar palsy does not therefore occur in elbow dislocations and fractures with backward or with backward and outward displacement Like contusion nerve injuries these traction injuries are often incomplete and the lesion is no more than a temporary loss of conductivity. Sometimes there is axonotmesis, with spontaneous recovery only after six or twelve months

Lateral popliteal paralysis—A similar traction nerve injury may complicate severe varus strains of the knee when the structures on the lateral side of the joint are torn or stretched The external lateral ligament is avulsed, with or without the styloid process of the fibula, and severe traction of the lateral popliteal nerve may cause paralysis. The prognosis of this injury is grave because of the very considerable length of nerve that may be damaged by traction Not infrequently there is intraneural fibrosis involving many inches of the nerve trunk so that spontaneous regeneration is impossible and operative treatment is impracticable.

Circumflex and other palsies in shoulder dislocations—In a series of 571 shoulder injuries there were 231 dislocations of the joint with 34 nerve lesions, and 340 fractures of the upper end of the humerus with no nerve lesions[1] The nerve injury is obviously due to traction, and the gross displacement of a dislocation is the most dangerous bone injury The head of the humerus is usually dislocated forwards The circumflex nerve and the posterior cord of the plexus, winding from the back of the axilla to the outer side of the arm, are therefore most frequently involved Less commonly there is paralysis of the ulnar nerve, of the musculospiral nerve, or of the other cords of the plexus. The paralysis usually recovers spontaneously.

The trunks of the brachial plexus or the nerve roots themselves may be stretched or even avulsed by traction injuries in which the arm is pulled away from the trunk. or the head is forcibly flexed to one side When this injury is sustained during delivery of a child, and one of the two common birth palsies results—namely, Erb Duchenne palsy involving the outer trunk or Klumpke palsy involving the inner trunk—the rule that traction nerve lesions usually recover spontaneously still holds true On the other hand, the more violent injuries of adult life may actually avulse the nerve roots from the cord and the prognosis is then hopeless.

Laceration of nerve—*Median nerve*—Median nerve paralysis in supracondylar fractures is usually due to simple nerve contusion, but occasionally the injury is more severe and the nerve is severed over the sharp lower margin of the main fragment of the humerus Spontaneous regeneration is then more difficult because there is actual loss of continuity of the nerve trunk The surgeon's suspicions may be aroused by the degree of displacement of the fracture, the sharpness of the proximal fragment, the completeness of paralysis or the delay in recovery. In such a case he should not wait for more than about three months If there is still no sign of recovery the nerve should be explored[2]

Musculospiral nerve—The musculospiral nerve in the middle third of the humerus is also vulnerable The nerve lies in a groove without muscular protection and in actual contact with bone All musculospiral nerve lesions in fractures of the humeral shaft should be regarded with suspicion Although the usual injury is a lesion in continuity which recovers spontaneously within three or four months, division of the nerve must be suspected when there is displacement of sharp fragments with complete paralysis which does not recover quickly

[1] Watson-Jones, R "Fractures in the Region of the Shoulder Joint" *Proc Roy Soc Med* (Section of Orthopædics), 1936, 29, 1061

[2] Platt, H, and Bristow, W R "Remote Results of Operations for Injuries of Peripheral Nerves (mainly Gunshot Injuries)" *Brit J Surg*, 1923, 11, 535

2. FRACTURES WITH SECONDARY NERVE INJURIES

Friction nerve lesions—Passive joint stretching may cause friction neuritis of the ulnar nerve with paralysis due to axonotmesis A fracture or dislocation causes roughening of the floor of the post-condylar groove and, although no complication arises until forcible passive movements are practised, friction of the nerve on the roughened bone, day after day, causes secondary traumatic neuritis In other cases the nerve is bound down by periarticular adhesions and its normal mobility is restricted Even a single forcible flexion movement under anæsthesia may then damage the nerve and cause paralysis

Late compression nerve lesions—It is doubtful whether compression of a nerve by the callus of a uniting fracture ever causes paralysis Such cases have been described in the past, but the lesion was probably due to friction of the nerve over callus as the result of passive stretching Late compression of a nerve by scar tissue has been held to be responsible for failure of regeneration particularly, for example, in traction lesions of the brachial plexus [1 2] It is on the assumption that extraneural scarring might cause compression of the nerve that the operation of neurolysis has been recommended, but the significant damage is the intraneural fibrosis, and it is by no means certain that benefit is gained by simple neurolysis

Late compression by splints, plaster, crutches, tourniquets—*The lateral popliteal nerve* lies in a vulnerable situation where it winds round the neck of the fibula A tourniquet should never be applied at this level, because it is so liable to cause paralysis The nerve may be compressed by splints, plaster or strapping, even the pressure of a simple bandage or viscopaste dressing may cause paralysis Special care must be taken, when applying an unpadded plaster cast, to protect the nerve by means of a pad of felt Moreover the upper margin of a below-knee plaster cast should be cut away to a level below the neck of the fibula in order to prevent friction and compression of the nerve during movement of the knee *The musculospiral nerve* is injured in a similar way if a full arm plaster extends no higher than three or four inches above the elbow. When the limb hangs by the side, the upper margin of the cast digs into the back of the arm and compresses the nerve against the humerus If compression and friction continue, paralysis supervenes within a few weeks There is much greater safety if the plaster is carried to a higher level, just below the axilla *Crutch paralysis*—Inadequate padding of axillary crutches, particularly when the crutches are too long, may cause musculospiral paralysis by compression of the nerve in the lower part of the axilla Padding with sorbo rubber or wool, and care to see that the crutch is not so long as to be driven up into the axilla, are sufficient to prevent this complication *Tourniquet paralysis*—Any form of rubber or inelastic tourniquet is dangerous in the upper limb Apart from the risk of gangrene (p 121), there is a very high incidence of musculospiral paralysis, and sometimes of median or ulnar paralysis. The flat rubber Esmarch tourniquet is little safer than other types. Only a pneumatic tourniquet with pressure controlled at 300 mm should be used

[1] Seddon, H J "Brachial Plexus Injuries" Editorial *J Bone Joint Surg*, 1949, **31-B**, 3
[2] Barnes, R "Traction Injuries of the Brachial Plexus" *J Bone Joint Surg*, 1949, **31-B**, 10

3. FRACTURES WITH DELAYED NERVE INJURIES

Late ulnar palsy—If in a child, a fracture of the lateral condyle of the humerus is not reduced accurately, so that the fragment is left rotated with no contact between the fractured surfaces, it fails to unite and increasing cubitus valgus deformity develops (Figs 240-241) Ultimately there may be 40° or 50° of deformity and the ulnar nerve is stretched round the inner side of the joint The deformity develops so gradually, and there is normally such free mobility of the nerve, that paralysis seldom occurs until adult life when it is precipitated by strenuous exercise Since the nerve lies behind the elbow it is stretched by flexion movement, but when there is cubitus valgus it is already stretched to its normal limit by the deformity The further stretching sustained during hard work causes paralysis ten or





FIG. 240 FIG. 241

Cubitus valgus following non-union of external condyle fracture in childhood. Stretching of the ulnar nerve round the inner side of the joint causes delayed neuritis and paralysis about ten years after the bone injury.

twenty years after the original bone injury The symptoms are completely relieved if the nerve is transposed to the front of the joint so that tension is relieved

Recurrent dislocation of the ulnar nerve—An injury to the internal epicondyle of the humerus may tear or stretch the fibrous roof of the post-condylar groove, or it may distort the bone so that the groove is unduly shallow. The ulnar nerve then slips forwards over the epicondyle with each flexion movement of the joint, and jerks back into its groove when the joint is extended Frictional neuritis due to the recurrent dislocation may cause ulnar paralysis, developing several years after the initial injury.

Tardy median palsy—Median paralysis may develop, from twenty to fifty years after fracture of the carpal bones, from compression of the nerve

where it lies in a confined space beneath the anterior carpal ligament by displaced bone fragments or the osteophytes of hypertrophic arthritis [1,2] The neuritis is of ischæmic origin and the paralysis recovers if compression is relieved by division of the anterior carpal ligament.

MEDIAN PARALYSIS

Etiology—Primary neuritis may be due to contusion of the nerve in dislocations of the carpal lunate bone and in supracondylar fractures of the humerus, and sometimes a traction injury is sustained in dislocations of the shoulder The nerve is occasionally severed at the elbow by supracondylar fractures [3] It may sustain traction injury in consequence of passive stretching of the stiff elbow In Volkmann's ischæmic contracture there is very often median paralysis due to loss of blood supply which may cause intraneural fibrosis over the whole length of the nerve trunk from elbow to wrist In the carpal tunnel there may be acute compression by displaced bone or by hæmorrhage in the palm, slow compression by the osteophytes of traumatic arthritis, or slow compression by occupational strain and pathological thickening of the anterior carpal ligament [4]

Clinical features—There is loss of sensation in the "three and a half finger area" (Fig 242) Patients often sustain cigarette burns of the anæsthetic fingers, and the loss of sensation of the index finger and thumb constitutes a serious disability. The thenar eminence is flattened from wasting and paralysis of the abductor and opponens pollicis, and the thumb tends to fall to the side of the hand ("ape-thumb deformity") When the nerve is injured at the elbow there is also paralysis of the pronator teres, flexor carpi radialis, flexor pollicis longus, and part of the flexor digitorum profundus But for the fact that flexion of the terminal joint of the thumb is impossible, the functional loss due to paralysis of these muscles can be masked

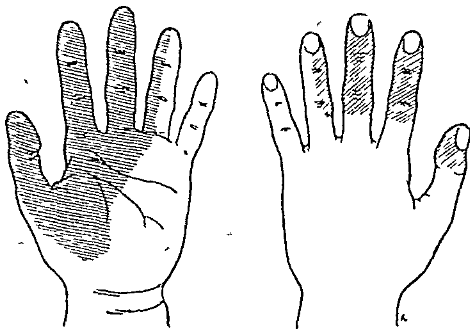


Fig 242
Area of anæsthesia in complete median paralysis

Treatment—Spontaneous recovery nearly always occurs within a few weeks in acute injuries of the wrist, and within a few months in injuries of the elbow and shoulder. It is usually unnecessary to explore the nerve, and the only treatment needed is galvanic stimulation of the affected muscles, with a small splint to hold the thumb in opposition and prevent stretching of the thenar muscles Caution is necessary, however, in supracondylar fractures of the humerus, particularly when the small distal fragment is displaced far backwards Complete division of the median nerve sometimes accompanies rupture of the brachial artery in supracondylar fractures

[1] Zachary, R B *Surg Gynec Obstet*, 1945, 81, 213
[2] Watson-Jones, R *J Bone Joint Surg*, 1949, 31-B, 560 (with bibliography)
[3] Platt H "Peripheral Nerve Complications of Fractures" *J Bone Joint Surg*, 1928, 10, 403
[4] Watson-Jones, R "Leri's Pleonosteosis, Carpal Tunnel Compression of the Median Nerve, and Morton's Metatarsalgia" *J Bone Joint Surg*, 1949, 31-B, 560

ULNAR PARALYSIS

Etiology—Ulnar paralysis seldom complicates injuries of the shoulder or wrist, but traction lesions are common in injuries of the elbow. It may occur in avulsion of the internal epicondyle, subluxation of the elbow with inclusion of the epicondyle, outward dislocation of the elbow, supracondylar fracture with outward displacement, supracondylar fracture with forward displacement and forward dislocation with fracture of the olecranon (Figs 235-239) Complete division of the nerve is practically unknown but severe stretching is possible, and it may even be caught between the articular surfaces of the elbow on its inner side, together with the displaced epicondyle Secondary neuritis may follow passive stretching or manipulation of the joint. Delayed neuritis occurs in external condyle fractures and recurrent dislocation of the nerve.

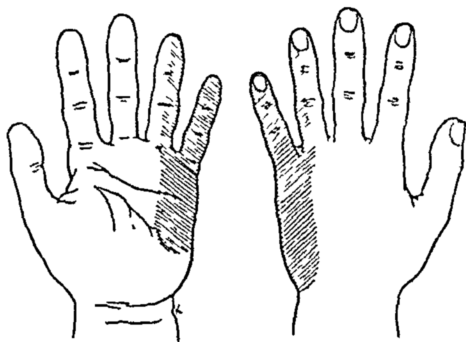


Fig. 243

Area of anæsthesia in complete ulnar paralysis.

Clinical features—In a complete lesion there is anæsthesia of the one and a half finger area (Fig. 243), and paralysis of the interosseous muscles, the flexor pollicis brevis and adductors of the thumb Paralysis of the lumbricales and interossei which normally flex the metacarpo-phalangeal joints and extend the interphalangeal joints causes the typical claw hand.

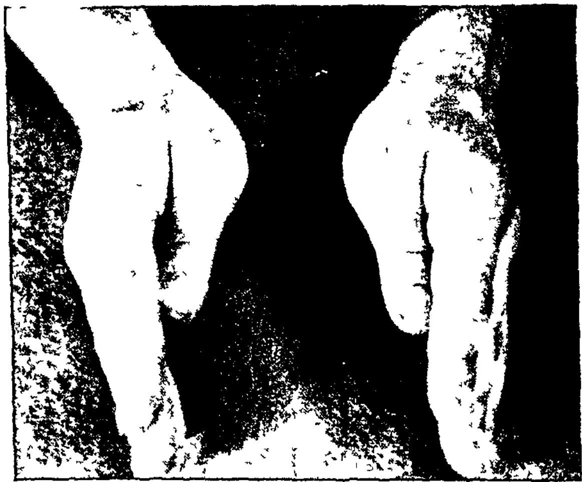


Fig 244



Fig 245

Incomplete ulnar paralysis Note the slight wasting of the first dorsal interosseous muscle of the left hand (Fig 244) The patient cannot keep his thumb flat (Froment's sign) This is the most reliable test for ulnar paresis (Fig 245)

Very often the lesion is incomplete In mild cases of traumatic neuritis there is no more than tingling in the fifth finger There may be a localised patch of anæsthesia or slight wasting or weakness of the intrinsic muscles of the hand (Fig 244) The classical test for muscle paresis is to estimate the power of gripping a sheet of paper between the sides of the fingers This test is unreliable It is better to ask the patient to grip a piece of wood or cardboard with the two thumbs and try to hold them flat (Fig 245).

The least trace of weakness of the adductor pollicis and short muscles inserted into the proximal phalanx is at once obvious, the patient cannot prevent the interphalangeal joint from flexing (Froment's sign)

Treatment—Conservative treatment is usually successful and recovery may be expected within six or twelve months Clawing and contracture of the fingers should be prevented by gentle stretching, carried out by the patient himself Passive stretching, forcible movements and manipulation of the elbow must be avoided If after epicondylar or supracondylar fractures there is irregularity and distortion of the postcondylar groove, anterior transposition of the nerve may be advisable[1] This is also necessary in delayed ulnar palsy due to cubitus valgus and in recurrent dislocation of the nerve.

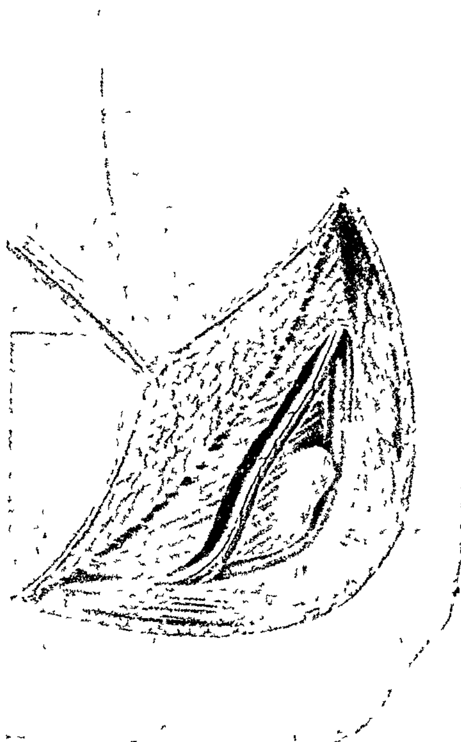


Fig 246
Anterior transposition of the ulnar nerve

Anterior transposition of the ulnar nerve—The nerve is dissected out in the lower two inches of the arm and the upper two inches of the forearm The branch to the elbow joint is divided but muscular branches are preserved The deep fascia and the superficial fibres of the muscles over the front of the inner condyle of the humerus are divided sufficiently to bury the nerve The internal intermuscular septum must be divided where it is crossed by the nerve in its new track, because otherwise there will be continued compression of the nerve at the level at which it rides over the sharp edge of the septum (Fig 246) The aponeurosis of the flexor muscles is then lightly sutured over the nerve and the wound is closed[2]

RADIAL (MUSCULO-SPIRAL) PARALYSIS

Etiology—The posterior interosseous nerve usually escapes in fractures of the neck and head of the radius, but the radial nerve may be injured in supracondylar fractures, fractures of the shaft of the humerus and dislocations of the shoulder The pressure of inadequately padded crutches may cause transitory paralysis

Clinical features—There is complete wrist drop due to paralysis of the extensors of the wrist, thumb and metacarpo-phalangeal joints of the fingers The interphalangeal joints can still be extended by the interosseous and lumbrical muscles The triceps muscle escapes because the branches that supply this muscle arise in the axilla The sensory loss is unimportant and is confined to a small area on the radial side of the dorsum of the hand.

[1] Platt, H "The Operative Treatment of Traumatic Ulnar Neuritis at the Elbow" *Surg Gynec Obstet*, 1928, 47, 822

[2] Learmonth, J R "Technique for Transplanting Ulnar Nerve." *Surg Gynec Obstet* 1942, 75, 792

Treatment—The prognosis is very good indeed. Simple lesions due to contusion always recover, and even when the nerve is completely severed and is sutured, permanent paralysis is exceptional In some cases of badly infected compound fracture of the humerus where nerve suture has had to be delayed for six or twelve months, and where at operation the nerve trunk was found reduced to a paltry strand, suture has been rewarded by complete recovery An attempt should always be made to suture the nerve, however difficult it may be Stretching of the paralysed muscles should be prevented by supporting the wrist in moderate dorsiflexion on a short cock-up splint or plaster cast A special splint may be used with elastic strips on the backs of the fingers to assist extension movement,[1] *but a full-length cock-up splint or plaster which immobilises the fingers in extension should never be used.* Such a splint often causes permanent stiffness of the fingers, which is a much worse disability than permanent paralysis. paralysis can be treated successfully by transplantation of muscle, whereas there is no treatment that will cure the crippling of permanent finger stiffness



FIGS 247, 248
Result of tendon transplantation for irrecoverable radial paralysis in compound fracture of the humerus The pronator teres, flexor carpi radialis and flexor carpi ulnaris transplanted to the extensors of the wrist, thumb and fingers Note that it is not possible to gain full extension of the fingers and wrist simultaneously It is therefore better to leave one wrist flexor.

Tendon transplantation for radial paralysis—For the few cases of irreparable nerve injury in which paralysis is permanent many excellent tendon transplantations are available In 1922 Robert Jones advised transplanting the pronator teres to the radial extensors of the wrist, the flexor carpi radialis to the extensors of the thumb and index finger, and the flexor carpi ulnaris to the extensors of the other three fingers The palmaris longus may also be transplanted to the abductor pollicis longus. But as Zachary [2] pointed out in 1947, if both wrist flexors are transplanted the wrist tends to dorsiflex so much that the metacarpo-phalangeal joints cannot be extended fully (Fig 247) It is better to leave the flexor carpi radialis as a flexor of the wrist and transfer the flexor carpi ulnaris to all extensors of the fingers and thumb

Transplantation for posterior interosseous nerve paralysis—If the lesion is not a paralysis of the radial nerve but of the posterior interosseous nerve (due, for example, to operative injury in the treatment of dislocations of the head of the radius, or fractures of the upper third of the shaft of the radius), the radial extensors of the wrist are not paralysed and one of them may be transferred to the extensors of the fingers and long extensor of the thumb. The palmaris longus may also be transferred to the abductor pollicis longus

[1] Highet, W B "Splintage of Peripheral Nerve Injuries" *Lancet*, 1942, 1, 555
[2] Zachary, R B "Tendon Transplantation for Radial Paralysis" *Brit J Surg*, 1946, 33, 358

CIRCUMFLEX PARALYSIS

Etiology—More than 5 per cent of shoulder dislocations are complicated by circumflex palsy The proximity of the nerve to the neck of the humerus, its limited mobility, and its course from the back of the axilla to the outer side of the humerus explain its vulnerability

Clinical features—The area of sensory loss is unimportant but there is complete paralysis of the deltoid This can be tested on the first day of injury, without actually moving the shoulder, by palpating the muscle with the fingers of one hand and asking the patient to attempt gentle abduction against the resistance of the other hand which is placed over the elbow Circumflex nerve lesions are to be distinguished from injuries to the posterior cord of the plexus (where there are the clinical features of combined circumflex and musculo-spiral paralysis), and from injuries to the outer trunk of the plexus (paralysis of the deltoid and the biceps)

Treatment—Stretching of the muscle should be prevented by supporting the limb in an abduction frame. If this is applied within a few days of injury, great care must be taken to prevent redislocation by bandaging the frame securely to the shoulder and trunk Some surgeons defer the application of the frame for three or four weeks The splint is worn day and night and the arm is never lowered below the right angle until recovery is sufficient to allow the patient to raise the limb actively from the frame Recovery is usually complete in two to six months During a five-year period I saw fifteen cases of circumflex palsy in dislocations of the shoulder of which ten recovered within six months, three within twelve months, and two were permanently paralysed [1]

SCIATIC PARALYSIS

Sciatic palsy in fracture-dislocation of the hip—Sciatic palsy occurs as a complication in about 7 per cent of traumatic dislocations of the hip The bone injury is nearly always a fracture-dislocation with tilting of a large acetabular fragment which impales the nerve in the sciatic notch (Fig 249) This association of injuries—dislocation of the hip, displacement of an acetabular fragment, and sciatic palsy—calls for prompt surgical treatment.[2] The dislocation should, of course, be reduced at once by manipulation, but if the acetabular fragment is not replaced accurately, it should be exposed It will be found that the nerve is impaled by the bone fragment and that compression of the nerve trunk is relieved only when the fragment is replaced

Occasionally the sciatic paralysis is complete In a complete lesion there is paralysis of all muscles below the knee, and anæsthesia below the knee except in the area supplied by the long saphenous nerve on the inner side of the leg Trophic lesions and traumatic ulceration often develop, especially when the foot is not only insensitive but is also subject to the abnormal pressure of pes cavus and rigid clawing of the toes which cause undue prominence of the metatarsal heads in the sole If these deformities are prevented by early assisted movements, trophic ulceration is usually avoided Even if deformity has already developed, and trophic ulceration

[1] Watson-Jones, R *Proc Roy Soc Med* (Section of Orthopædics), 1936, 29, 24, 1062
[2] Armstrong, J R "Traumatic Dislocation of the Hip" *J Bone Joint Surg*, 1948, 30-B, 437 and 441

has supervened, amputation is still not inevitable; correction of the deformity may allow healing of the ulcers despite anæsthesia of the sole.

Incomplete sciatic palsy—More frequently, sciatic paralysis associated with dislocation of the hip joint is incomplete, and the lateral popliteal division is involved. There is drop foot and paralysis of the peronei, but



Fig 249

Fig 250

Displacement of an acetabular fragment in fracture-dislocations of the hip joint often causes sciatic paralysis. If displacement of the fragment is not corrected by manipulative reduction of the dislocation, operative replacement is urgently needed

(*Treated in the Royal Air Force Orthopædic Service by Mr J C Armstrong*)

sensation of the sole of the foot is normal. A toe-elevating spring should be fitted, or an inside iron with drop foot stop and outside T-strap. If the paralysis does not recover, permanent use of a leg iron or toe-elevating spring can be avoided by operative stabilisation of the foot by the Lambrinudi technique[1,2]. The subastragaloid and mid-tarsal joints are fused, but with

[1] Lambrinudi, C "New Operation on Drop Foot" *Brit J Surg*, 1927, **15**, 193

[2] Lambrinudi, C "A Method of Correcting Equinus Deformities at the Subastragaloid Joint" *Proc Roy Soc Med* (Section of Orthopædics), 1933, **26**, 788

a wedge cut, base forwards, at the subastragaloid level, so that the foot cannot drop more than about 25° below the right angle

LATERAL POPLITEAL PARALYSIS

Etiology—The nerve may be stretched when there is rupture of the external lateral ligament, avulsion of the styloid process of the fibula, or dislocation of the knee joint [1 2] Secondary lesions arise from compression of the nerve against the neck of the fibula

Clinical features—There is paralysis of the anterior tibial and peroneal muscles, and anæsthesia of the outer aspect of the leg and dorsum of the foot Mild contusion and compression injuries recover spontaneously within a few months Stretching of the paralysed muscles must be prevented by a plaster cast with the foot fully dorsiflexed and in slight eversion After weight-bearing is resumed, an iron with a drop foot stop should be worn until recovery is complete In traction injuries of the lateral popliteal nerve the prognosis is much more grave Many inches of the nerve may suffer intraneural fibrosis, and although it is a lesion in continuity (axonotmesis) the damage may be so extensive that regeneration cannot occur Operative resection and suture is not possible and the palliative measure of stabilisation of the foot by the Lambrinudi technique is the only alternative to the permanent use of an inside iron and drop foot stop or toe-elevating spring.

[1] Platt, H "Ext Popliteal Nerve Involvement in Fractures of Fibula" *J Bone Joint Surg*, 1928, **10**, 412
[2] Watson-Jones, R "Styloid Process of Fibula in Knee Joint with Peroneal Palsy" *J Bone Joint Surg*, 1931, **13**, 2, 258

CHAPTER VIII

CLINICAL AND RADIOGRAPHIC DIAGNOSIS

CLINICAL DIAGNOSIS

In many bone and joint injuries the clinical features are obvious and simple inspection reveals the classical signs of local swelling, ecchymosis and deformity No attempt should be made to elicit the other classical sign, namely, crepitus due to grating of the fragments on each other Manipulation for the purpose of diagnosis is unnecessary it causes unnecessary pain and may be responsible for damage to the blood vessels or nerves , the diagnosis can be usually made on inspection and palpation alone , *there is no necessity to move the limb at all* The position and contour should be compared with the normal limb. When an elderly patient falls and injures the hip joint, slight external rotation deformity is sufficient to make a provisional diagnosis of fracture of the femoral neck even when there are no other clinical signs , flattening of the contour of the shoulder may disclose a dislocation of the joint , slight prominence of the lower end of the ulna shows a fracture of the radius ; and so on

Localised bone tenderness—The sign that needs emphasis because it is sometimes ignored is persistent local tenderness over one part of the bone In many impacted fractures, crack fractures and greenstick fractures, this may be the only clinical sign If there is localised bone tenderness a fracture should be assumed until it is disproved by radiographic examination. Fractures of the carpal scaphoid bone are often overlooked and prolonged or even permanent disability is caused by attributing tenderness over the radial side of the wrist to a simple sprain

Diagnosis of injury to soft parts—When it is established that there is a fracture, an equally important part of the clinical examination still remains : the limb must be examined for injury to the vessels and nerves , in fractures of the spine, careful neurological examination is essential , and in thoracic and pelvic injuries, visceral damage must be excluded In no case of injury to a bone should the surgeon allow his attention to be focussed on the particular injury to the exclusion of other possible injuries Errors often arise because surgeons apply the whole of their attention to the first injury they happen to see.

RADIOGRAPHIC DIAGNOSIS

Legal actions against surgeons who were responsible for the treatment of fractures have often been difficult to defend because no X-ray films had been taken Radiographs may show injury or displacement that is not evident on clinical examination Even when there is so obvious an injury as dislocation of the elbow joint, X-ray films will show whether or not there is also avulsion of the medial epicondyle with danger of inclusion of the



FIG 251



FIG. 252

Radiographs of the ankle joint (Fig 252) show backward dislocation and a posterior marginal fracture of the tibia but no fracture of the fibula This displacement could not arise without a fracture of the fibula The upper half of the fibula must be X-rayed (Fig 251)

fragment within the joint; they will show whether there is an associated fracture of the head of the radius needing operative excision. Radiographs of a dislocated shoulder may reveal fracture of the great tuberosity which is not recognisable on clinical signs and may alter the whole plan of treatment

Screening is unreliable and films of the best quality should always be taken. Diagnosis should not be attempted unless there is good contrast between bone and soft tissues, and unless the injured region is in the middle of the film so that the whole suspected area is covered Radiographs of fractures of long bones should always include the joint above or below, and preferably both A short film that covers only the shaft of a bone is



FIG 253

A surgeon accepted this film, and believed that the injury was a simple fracture of the ulna, needing no treatment other than the application of plaster



FIG 254

It is now obvious that the injury is not a simple fracture of the ulna There is also a dislocation of the head of the radius which must be reduced

often worthless because it may be impossible to know the direction of displacement. Moreover, in injuries of the forearm and leg, a fracture of the shaft of one bone may be accompanied by an injury to the other bone at an entirely different level If there is a fracture of the shaft of the tibia with over-riding, and no evidence in the film of injury to the fibula, the whole length of the fibula should be examined · there is probably a fracture of the neck of the bone Backward dislocation of the ankle joint may be accompanied by fracture of the upper shaft of the fibula (Figs. 251-252). An apparently isolated fracture of the upper shaft of the ulna is nearly always associated with subluxation or dislocation of the head of the radius and although there may be no clinical evidence of the radial displacement it is by far the most important part of the injury (Figs. 253-254). A fracture

of the lower shaft of the radius may be shown by radiographic examination to be part of a fracture-dislocation with displacement of the ulna, which indicates the likelihood of redisplacement of the radial fracture and the need for operative internal fixation Radiographic examination of every fracture shows the precise direction of displacement and the degree of manipulative strength that is required to reduce it From every point of view, failure to arrange radiographic examination of a suspected bone or joint injury is indefensible

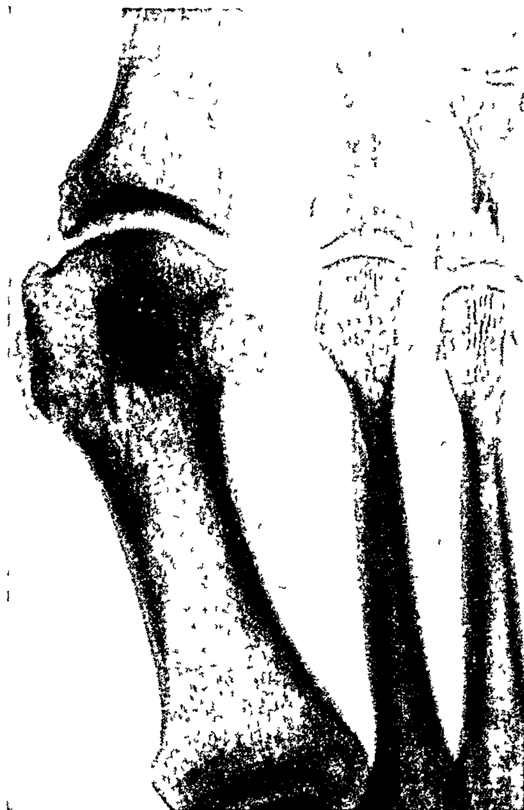


FIG 255

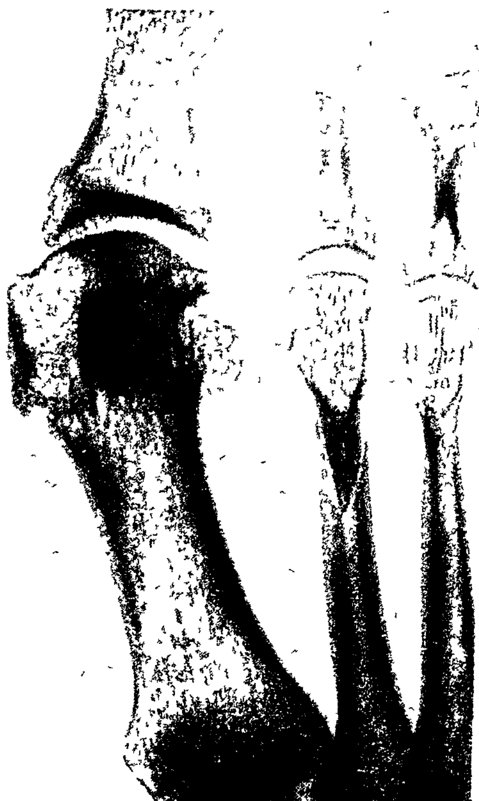


FIG 256

March fracture of the metatarsal is the clearest example of a fracture which when first sustained may be so fine a crack as to be not visible in X-ray films (Fig 255) But the clinical diagnosis is more reliable than the radiographic diagnosis There is a fracture even if radiographs fail to disclose it It becomes obvious in radiographs three weeks later (Fig 256)

It must be admitted, however, that the danger to-day is not so much that the *evidence of radiographic examination* will be neglected as that it will be exalted There is danger in believing that X-ray evidence of displacement of a bone is an absolute indication for correction of the displacement Many operations have been undertaken with the object not of improving function but of improving radiographic appearances It is an unhappy reflection that sometimes, particularly perhaps in the United States of America, surgeons have found it necessary to undertake operations solely with the object of improving radiographic appearances and thus defending themselves before a lay judge and jury in the event of an action being brought Let us hope that we may soon return to the day when interpretation of radiographs is accepted as the duty of radiologists and surgeons but not of laymen, when the use of X-rays is regarded as no more than one of a number of controls

of fracture treatment, and when functional results are recognised as being more important than radiographic appearances

Danger of undue reliance on X-rays—There is equal danger that the evidence of radiographic examination shall be allowed to outweigh the evidence of clinical examination which may be more reliable A crack fracture of bone is sometimes so fine that, for some days after injury, it cannot be distinguished radiographically from the normal lines of separation of trabeculæ The clinical signs of "march fracture" of a metatarsal bone may be obvious long before there is radiographic evidence (Figs 255-256). The clinical signs of a recent crack fracture of the carpal scaphoid bone



Fig 257
The arthrodesis seems to be well united and there is a suggestion of continuity of trabeculæ from one bone to the other indicating final consolidation

The fact is that this radiograph was taken in the operating theatre within a few minutes of completing the arthrodesis Union has not yet started, only accurate apposition of the bones and tight packing with cancellous chips gives an appearance of union and even of continuity of trabeculæ Radiographs are shadows, and they are often deceptive

are often more reliable than the radiographic appearances. Fatigue fractures of bone are often concealed during the first few weeks In these minor bone injuries radiographic evidence is very fallacious

Similarly, when attempts are made to assess the degree of union of a fracture, or of an arthrodesis, overlap of radiographic shadows may be misleading The hip joint is notoriously difficult in this respect, but there is sometimes equal difficulty in other joints, as, for example, in Figure 257 where arthrodesis of a knee joint appears to be consolidated within a few minutes of completing the operation !

Radiographs in two planes at right angles—It must always be recognised that a radiograph is no more than a shadow of bone and that shadows often distort and conceal. Even when a bone is angulated in one direction through



FIG. 258

The injury appears trivial The slight splintering of the malleoli looks to be unimportant It would appear that no special treatment is needed.



FIG 259

The lateral film, taken at the same time as Figure 258, shows a serious fracture-dislocation with backward displacement There must always be two views at right angles



FIG 260

The antero-posterior radiograph suggests that if there has been any fracture of neck of femur at all, it is impacted in perfect position The surgeon responsible for this case did not believe from this evidence that any special treatment was needed



An elderly patient stumbled and complained of pain in the hip. There was no definite physical sign of injury but the patient lay with the limb in external rotation The routine antero-posterior radiograph is seen in Figure 260. Can it be accepted that there is no serious injury to the hip ?

The l
fractur
serious
Two vi
the hip,

Raise flap to see Figure 261

90°, there is a plane in which the shadow shows an appearance of perfect alignment It is almost better to have no X-ray at all than a single film in one projection Without the benefit of any radiograph the surgeon does at least know that he must rely on clinical signs ; whereas with a radiograph his mind is dominated and he may imagine that what he sees in a single

…osition of the bones How fallacious this can be

…9

…at many surgeons who would almost be insulted …ey do not insist upon both antero-posterior and …ared to accept a single film of the hip and shoulder … it is by no means usual for the X-ray departments

A patient sustained an injury to the ankle joint. The antero - posterior radiograph seen in Figure 258 shows slight splintering of the malleoli but no evidence of complete fracture, of dislocation, or of any displacement Can this evidence be accepted ? Is the injury as trivial as it appears to be ?

Raise flap to see Figure 259

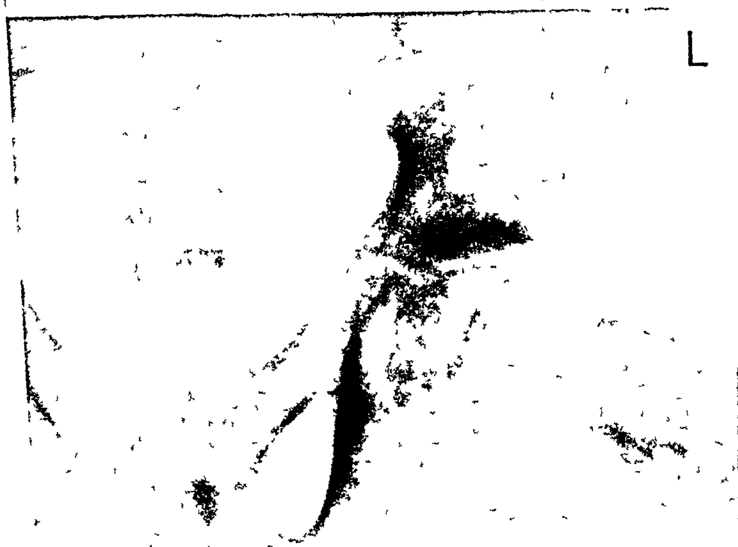


FIG 262

The early stages of slipping of the upper femoral epiphysis are shown only in lateral projections In the lithotomy position, with the hips flexed, abducted and externally rotated, early slipping is seen on the right (*By courtesy of Mr Hambly, London*)

of general hospitals to submit routinely more than a single projection of these two joints , and yet it is no less true of the hip than of any other joint that as much as 90° of displacement of bone fragments can be concealed in antero-posterior projections (Figs 260-261) Many traumatic dislocations of the hip joint have been left unreduced because, although the femoral head was pulled down to the level of the acetabulum, it remained *behind* the acetabulum but with fairly accurate overlay of shadows, so that in the antero-posterior view it appeared to be reduced Similarly the early stages of slipping of the upper femoral epiphysis are concealed in antero-posterior radiographs , lateral radiographs of both hips should be taken with the patient in the lithotomy position so that the joints are flexed, abducted and externally rotated (Fig 262)

In the shoulder joint one of the displacements most constantly left unreduced is forward angulation of fractures of the neck of the humerus, amounting even to 80° or 90°, which is concealed in antero-posterior views

This radiographic error explains long persistence of the use of a type of abduction frame in which the limb lay in the plane of the trunk, instead of in the neutral position 80° in front of the plane of the trunk

There is probably no bone or joint injury that has more often been overlooked than posterior dislocation of the shoulder The physical signs are usually masked by swelling , with the expert eye an abnormality may be noted in the antero-posterior radiograph but the appearances are by no means obvious Look at Figure 263. It is a radiograph of good quality , but is the diagnosis at once obvious ? Only when lateral films (" vertical " projections) are taken with the tube below the axilla and the casette above the shoulder, or alternatively with a curved casette in the axilla and the tube above the shoulder, is the degree of displacement disclosed (Fig. 264)

At least two radiographic projections at right angles are always necessary. Moreover the two films should be put to the light side by side, and examined simultaneously, the surgeon always thinking in terms of three dimensions Lateral radiographs of the hip joint are no less important than antero-posterior radiographs Vertical projections of the shoulder joint should be taken routinely

Radiographs in three planes—It is often assumed that if radiographs are taken in two planes at right angles to each other, no bone injury or displacement can escape notice. This is erroneous A fracture may lie in a plane of such obliquity that the shadows of the fragments overlap accurately in both the classical positions The injury is disclosed only when a film is taken in the oblique axis This very often applies to fractures of the carpal scaphoid bone Fractures of the waist of the scaphoid, and of the tubercle of the scaphoid, may be masked in strict antero-posterior and lateral views even if the radiographs are examined stereoscopically Furthermore a fracture of the waist of the scaphoid which has been immobilised may appear to be united when examined only in antero-posterior and lateral views, and yet an oblique view shows that union is unsound It should be a routine practice in radiography of the scaphoid bone to take at least three views

In the lumbar spine the neural arches and articular facets are concealed in routine antero-posterior and lateral views but are shown clearly in the oblique projection In this view the shadow of the neural arch may be compared to the outline of a Scotch terrier—the trunk of the dog is the lamina, the ear the ascending articular process, the nose the transverse process and the front paw the descending articular process (Fig 266) This projection is of particular value in recognising spondylolysis and spondylolisthesis. If the terrier appears to be wearing a collar, the spine is not normal—there is spondylolysis with a fibrous defect between the lamina and descending articular process on the one hand and the ascending articular process, transverse process and vertebral body in the other—the area of bone described as the " pars interarticularis " (Fig 267) If the dog is beheaded, so that there is an actual gap at this level, the lesion is a spondylolisthesis (Fig 268) Not only does this oblique projection make it easy to recognise the pathological anatomy of spondylolysis and spondylolisthesis, but it may demonstrate fractures of the neural arch and the articular facets which are concealed in routine radiographs

Repeated radiographic examination after an interval—If a radiograph fails to show evidence of a fracture which is suspected strongly on clinical



FIG 263

Antero-posterior radiograph of the shoulder joint in the case reported below

A man aged 43 years was cycling and ran into the back of a lorry He struck his right shoulder Clinical examination appeared to show normal contours, but the coracoid process was a little more prominent than usual There was no other sign of abnormality except that the limb was held in internal rotation, and external rotation movement was resisted. Figure 263 shows the anterior-posterior radiograph. Can it be assumed that there is no fracture or dislocation ?

Raise flap to see Figures 264-265.



FIG. 266

Oblique projections of the spine display the laminæ and articular processes more clearly than classical antero-posterior and lateral projections The shadow of the neural arch resembles that of a Scotch terrier the ear is the ascending articular process, the nose is the transverse process, the front paw is the descending articular process



FIG 267

If the terrier is wearing a collar there is spondylolysis



FIG. 268

If the terrier is beheaded there is spondylolisthesis.

grounds the examination should be repeated after two or three weeks. If there is a fracture, movement of the fragments during the interval causes traumatic hyperæmic osteoporosis so that the inconspicuous crack becomes an obvious fracture. This is especially important in suspected fractures of the carpal scaphoid bone where the crack may be so fine that even good films do not demonstrate it (Figs. 269-270) Similarly, in suspected march fractures of the metatarsal bones, radiographs taken within a day or two of the onset of symptoms often fail to show the fracture; whereas after two or three weeks the line of fracture is obvious and there is also subperiosteal callus formation (Figs. 255-256).



FIG. 269

There is no definite radiographic evidence of fracture of the scaphoid bone

A patient sustained a wrist injury and the clinical features suggested a fracture of the scaphoid. Figure 269 is the antero-posterior radiograph, lateral and oblique views showed no abnormality Is there a fracture of the scaphoid ? Can a fracture be excluded on this radiographic evidence ?

Raise flap to see Figure 270

Slight crush fractures of a vertebral body may undergo spontaneous reduction by alteration in the posture of the patient The injury is produced by flexion of the spine; if the spine is then fully extended the displacement is sometimes reduced so perfectly that no radiographic evidence remains Unless the spine is immobilised in extension the damaged vertebra will slowly collapse and be compressed by weight-bearing strain A second radiographic examination, ten to fourteen days after the first, will show the early stages of this recurrent wedging and confirm the diagnosis which could not be established by the original examination

Radiographs in different positions of the joint—Certain bone and joint injuries are concealed in routine radiographs and disclosed only when radiographs are taken with the joint in a position of strain

Sprains and subluxations of the ankle joint—For many years it was believed that dislocation of the ankle joint, unaccompanied by fracture, was exceedingly rare It was also believed that sprain of the ankle joint was a minor injury that could be treated safely by strapping support and early mobilisation, this view being held despite the conviction of many patients that a sprain was more serious than a fracture Actually, dislocation of the ankle joint occurs frequently but the displacement is momentary and it undergoes spontaneous reduction, the clinical signs then suggest a simple sprain and radiographs show no evidence of bone or joint injury (Fig 271). Only radiographs taken with the foot held in the fully inverted position

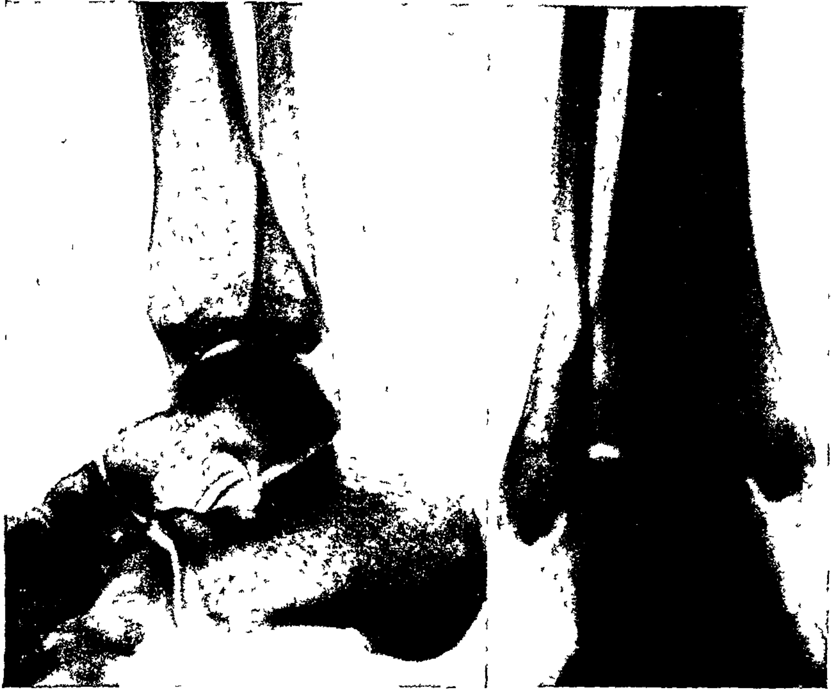


Fig. 271

Severe sprain of the ankle. There is no radiographic evidence of bone or joint injury



Fig 272

Repeated examination with the foot in maximum inversion shows that the "sprain" is actually a complete dislocation The external lateral ligament is completely ruptured

show tilting of the talus within the tibio-fibular mortice, thus proving momentary dislocation of the joint and complete rupture of the lateral ligament (Fig 272) This injury necessitates immobilisation in plaster for not less than eight or ten weeks If it is treated as a simple sprain by early mobilisation the ligament fails to unite, the joint remains unstable recurrent dislocation develops, and the talus subluxates every time the foot is inverted. The patient dare not walk freely over rough, irregular surfaces and the foot constantly gives way, he may fall to the ground In former years, when routine radiographic examination was relied upon, the explanation was obscure and it was not surprising that patients who travelled from one doctor to another, from osteopaths to bonesetters, and from manipulative surgeons to naturopaths, who were treated by massage, electrotherapy and manipulation, and still gained no relief from their disability, believed that "sprain" of the ankle was a most serious injury

The diagnosis of momentary subluxation, and the sequel of recurrent dislocation of the ankle joint, were described in the first edition of this book in 1940 and since that time the technique of radiographic examination of the inverted foot has been accepted The frequency of the injury is now recognised Rowland Hughes [1] pointed out that in 50 per cent of sprained ankles it was easy to demonstrate slight tilting of the talus, and in 20 per cent of injuries there was considerable tilt which amounted to subluxation of the joint A clear distinction must therefore be drawn between sprains due to stretching of fibres of the anterior fasciculus of the lateral ligament which may be treated safely by novocaine injection, massage, or simple elastic support, and momentary dislocations with rupture of the middle fasciculus and tilting of the talus in which complete immobilisation in plaster is necessary The radiographic test may not be reliable unless local novocaine anæsthesia is used because otherwise the patient resists the painful movement by strong peroneal spasm Moreover, the surgeon should hold the foot himself and not reply on radiographers who are afraid of causing further injury and may fail to demonstrate the displacement because they do not invert the foot strongly enough Finally, whatever grip the surgeon may choose to employ, he must invert the heel and not simply adduct the forefoot

Rupture of the medial ligament of the ankle joint—In the same way that rupture of the lateral ligament is concealed unless radiographs are taken of the inverted foot, rupture of the medial ligament may be concealed unless radiographs are taken of the everted foot This routine is important in undisplaced subperiosteal fractures of the lateral malleolus if it is proposed to adopt the treatment of novocaine injection and immediate unprotected weight-bearing Such treatment may be safe if the fracture is the isolated injury it appears to be, but routine radiographs show no distinction between isolated fractures of the lateral malleolus and fractures of the malleolus with rupture of the medial ligament and potential outward dislocation of the foot Before unprotected weight-bearing is permitted, radiographs should be taken with the foot held in full eversion, preferably after novocaine injection of both malleolus and ligament

Inferior tibio-fibular diastasis—Even when there is no fracture it is possible for the talus to subluxate outwards if rupture of the medial ligament of the

[1] Hughes, J R "Sprains and Subluxations of the Ankle Joint" *Proc Roy Soc Med*, 1942, 35, 765

ankle joint is accompanied by rupture of the inferior tibio-fibular ligament. This subluxation is no less important a source of permanent disability than Pott-Dupuytren fracture-dislocations of the joint; the displacement must be reduced; the joint must be properly immobilised. Every severely sprained ankle should therefore be X-rayed with the foot held both in full inversion and full eversion

Rupture of the inferior tibio-fibular ligament with diastasis of the joint may also occur as a complication of abduction fracture-dislocations of the ankle joint. It is difficult to prevent permanent disability because redisplacement may occur despite the protection of a carefully moulded plaster cast The diastasis is sometimes concealed in routine radiographs because the joint is obscured by the overlapping shadow of the tibia. It is therefore advisable to take radiographs with the foot held in full eversion. If the gap on the inner side of the joint, between talus and medial malleolus, is greater than can be accounted for by displacement of the lateral malleolus, diastasis of the inferior tibio-fibular joint is proved and steps must be taken accordingly

Subluxation of the cervical spine—A suspected fracture or dislocation of the cervical spine which is not shown in routine radiographs may be disclosed by repeating the lateral examination with the spine in a moderate degree of flexion This routine was of great importance to the patient whose radiographs are shown in Figures 273-274 He was sitting in a vehicle which stopped suddenly so that his head was jerked sharply forwards There was no direct injury, but he complained of severe pain in the neck and down both arms Radiographic examination showed no bone or joint injury and the history of accident was vague After six weeks he was accused of malingering, his compensation was stopped and he was told to resume heavy work. The distribution of pain suggested a possible subluxation of the cervical spine with root pressure, and it was easy to confirm this by repeating the radiographic examination with the spine in flexion (Fig 274). There is obviously an incomplete dislocation of the interarticular and intervertebral joints which appears only in the flexed position Immobilisation in plaster was necessary for ten weeks in order to allow tightening of the damaged ligaments, the man ultimately returned to full work despite the arthritic change

Hyperextension subluxations of the cervical spine—It has been pointed out recently by Barnes,[1] and by Taylor and Blackwood,[2] that many cervical injuries, especially in elderly patients with stiff and arthritic joints, are due to hyperextension strain with momentary backward subluxation or dislocation which is reduced spontaneously. This accounts for many, if not perhaps all, cases of cervical injury in which radiographic examination appears to show no evidence of bone or joint injury and yet there is paraplegia due to cord injury It is evident, therefore, that even if routine antero-posterior and lateral radiographs of the spine show no evidence of bone or joint injury there may still be a fracture of the neural arch, a reduced subluxation, or even a complete dislocation of intervertebral and interarticular joints, which may be disclosed in lateral projections taken with the spine cautiously flexed or cautiously extended.

[1] Barnes, R "Paraplegia in Cervical Spine Injuries" *J Bone Joint Surg*, 1948, 30-B, 234
[2] Taylor, A R, and Blackwood, W "Paraplegia in Hyperextension Cervical Injuries with Normal Radiographic Appearances" *J Bone Joint Surg*, 1948, 30-B, 245

Radiographs of the shoulder in different positions of rotation—Radiographic examination of the shoulder joint in different rotational positions displays different profiles of the head of the humerus and may reveal pathological changes which are concealed in the classical antero-posterior projection taken with the limb in neutral rotation. The insertion of the supraspinatus tendon is seen on top of the great tuberosity when the limb is in full lateral rotation; the insertion of the teres minor is in profile when the limb is in full medial rotation Routine radiographic examination should include antero-posterior projections in lateral rotation, neutral rotation, and medial rotation, as well as lateral projections with the tube at the side of the chest and the casette above the acromio-clavicular region which shows the insertion of the subscapularis, otherwise fractures of the tuberosities and calcification in the tendons around the joint may be overlooked. The best example of unrecognised bone injury—unrecognised because antero-posterior radiographs alone were relied upon—is compression fracture of the head of the humerus in recurrent dislocation of the shoulder joint This compression, which is due to repeated impact of the humerus against the anterior margin of the glenoid, is concealed in antero-posterior radiographs in the neutral and laterally rotated positions (Figs 275-276) and yet it is shown quite clearly in 80 per cent of cases if the film is taken with the limb rotated medially through 60° or 70° (Fig 277) [1]



Fig 273

The classical antero-posterior and lateral views of the cervical spine show no evidence of bone or joint injury

After a flexion strain of the neck, the patient whose radiograph is shown in Figure 273 complained of pain radiating down both arms and of tingling in the fingers There is arthritis at the fifth cervical level shown in the lipping of the margins of the vertebral body

Is there evidence of injury? Can a dislocation or subluxation of the cervical spine be excluded?

Raise flap to see Figure 274

[1] Adams, J C "Recurrent Dislocation of the Shoulder" *J Bone Joint Surg*, 1948, **30-B, 26**



Fig. 275



Fig. 276

In a patient with recurrent dislocation of the shoulder joint, radiographs taken in neutral rotation (Fig. 275) and in external rotation (Fig. 276) show no evidence of abnormality in the head of the humerus (but see Fig. 277).



Fig. 277

The same patient whose radiographs of the shoulder joint in neutral and external rotation are shown in Figures 275 and 276 has, in fact, a very large compression defect in the head of the humerus. This defect is shown in profile only in radiographs taken with the joint internally rotated through 70°.

Axial views of the patella—In the knee joint, injuries are often concealed in routine radiographs In antero-posterior projections the shadow of the patella is overlapped by that of the femur, and the usual antero-posterior and lateral views really give only one projection of the patella, so that injuries are often overlooked This applies particularly to osteochondral fractures sustained in consequence of momentary lateral subluxations or dislocations of the bone[1] Axial projections should be taken, the joint being flexed and the ray directed through the patello-femoral joint in a vertical direction (Fig 278)



Fig 278

Axial view of the patella.

Antero-posterior radiographs of the knee joint in flexion—Antero-posterior and lateral views show only part of the curved surface of the femoral condyles in profile In many cases there may have been injury to the articular surface, or separation of a fragment of bone by the process of osteochondritis dissecans, involving some other part of the surface than that which happens



Fig 279



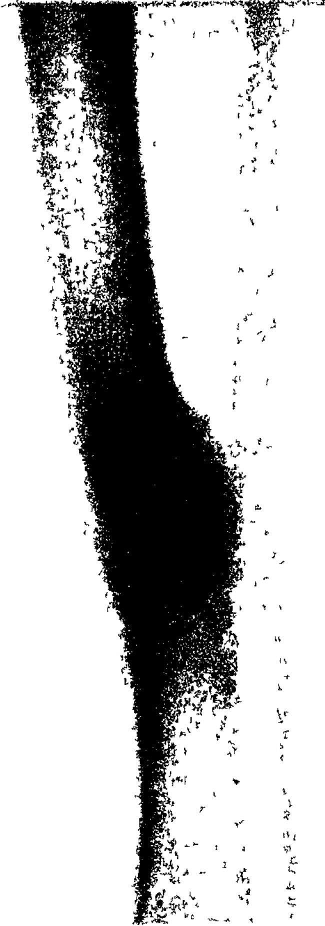
Fig 280

The clinical features in this patient suggested the likelihood of separation of a loose body by osteochondritis dissecans, but antero-posterior and lateral radiographs showed no abnormality (Fig 279) When the antero-posterior projection was repeated with the joint flexed 30°, so that a different part of the curved articular surface of the femoral condyles was brought into profile, the lesion was quite obvious (Fig 280).

to be in profile It is therefore wise to take antero-posterior projections of the flexed joint, the patient kneeling on the film first with his thigh at an angle of 30° to the table-top, then at 45°, and, if necessary, again at 60°.

[1] Coleman, H M "Recurrent Osteochondral Fracture of the Patella" *J Bone Joint Surg*, 1948, 30-B, 153.

DIAGNOSIS OF UNION OF A FRACTURE



is better expressed in the words "there is not yet radiographic evidence of consolidation"

Importance of radiographic evidence—In certain fractures it is important that clinical tests of union should be supplemented by radiographic evidence before splints or plaster are discarded In fractures of the carpal scaphoid bone no clinical test of union is dependable, and the surgeon must rely entirely on radiographic evidence In fractures of the shafts of the leg bones, clinical examination may appear to show firm union at a time when unprotected weight-bearing would be followed by yielding of the fracture This was a frequent cause of non-union and mal-union in former years when patients were discharged from hospital, often within six or eight weeks of injury, as soon as union appeared to be firm on clinical examination. In



FIG 283

In the antero-posterior radiograph union appears sound



FIG 284

The lateral radiograph shows established non-union

such fractures the diagnosis of union should be based on the combined evidence of clinical and radiographic signs

Radiographic evidence of union—Union of a fracture may be accepted when there is a continuous external bridge of callus joining the fragments, or when callus between the fragments is uniformly calcified and of a density approaching that of normal bone This evidence must be interpreted with care No film should be accepted unless it is correctly exposed, properly developed, and without fog, so that there is minimal soft-tissue shadow, a bright bone shadow, and clearly visible bone texture An under-exposed film with a heavy soft-tissue shadow may give an appearance of calcification of callus which is actually due to the overlying soft tissue (Figs 281-282) The possibility of overlap of bone shadows must also be excluded (Fig 257) A single radiograph is often misleading (Figs 283-284) Even two views at right angles may cause error if neither view coincides with

the plane of an obliquely placed fracture This nearly always applies to fractures of the carpal scaphoid bone and one or more oblique views must be taken routinely In other fractures it is often wise to supplement the traditional antero-posterior and lateral radiographs with oblique views, particularly in fractures of the shaft of the tibia

If there is doubt, the radiographic examination should be repeated with lateral strains applied to the fracture site In a fractured tibia, for example, a film is taken while the limb is held with varus strain and compared with one taken with valgus strain. Difference in the alignment of the fragments



Fig. 285

Old fracture of lateral condyle of humerus Is the union firm enough to prevent increasing valgus deformity or not ?



Fig 286

Same case as Figure 285, taken with forearm held in valgus position Union is unsound There will be increasing cubitus valgus and ultimately ulnar paralysis

in the two films, showing elastic springing of the fragments, proves that union is not sufficiently firm for unprotected weight-bearing It is often advisable to draw a line on the film in the axis of each fragment so that alignment can be judged more accurately

The most important check, in cases where radiographic evidence is inconclusive, is to insist on repeating the X-ray examination after an interval of three or four weeks This precaution is especially important in fractures of the carpal scaphoid bone, and in lower-limb fractures where it has been decided to discard the plaster and allow weight-bearing After an interval of three or four weeks, unsound union is shown by deossification and resorption of the callus, if the density of bone formation has increased despite lack of protection, union is sound

Radiographic evidence of consolidation—Final consolidation of a fracture is shown by the uniform and uninterrupted calcification of callus, the absorption and consolidation of external callus, and the development of continuous trabeculæ across the fracture site. The diagnosis may be confused, however, exactly like the earlier diagnosis of union, by overlapping bone shadows. Figure 285 shows an old fracture of the lateral condyle of the humerus in which it is difficult to be certain of the degree of union. Will this remain a stable elbow and continue to give good service, free from symptoms and from complications; or is it an unstable elbow which will develop increasing cubitus valgus deformity and ultimately lead to delayed ulnar palsy? A radiograph taken with the elbow held with the greatest possible valgus deviation of the forearm gives the answer (Fig. 286). The union is unsound.

THE DANGER OF X-RAYS TO SURGEONS

Many surgeons have wrecked their careers by fluoroscopic screening of fractures: dermatitis of the hands has made it impossible to scrub up; chronic ulceration has needed surgical excision and plastic repair; fingers have been amputated; cancer has developed.[1,2] This is not an idle threat. It is what actually happened to ninety-one surgeons who used fluoroscopy for reducing fractures.[3,4] The figure relates only to published cases in one locality and it does not include radiodermatitis from causes other than fracture treatment; it does not include milder cases of erythema, fissured nails, rough skin, slight telangiectasis, pigmentation, atrophy of the fat pads and hang-nail; it includes only severe cases of indurated board-like skin, acute and chronic ulceration, and cancer.

If a surgeon puts his hands in the direct beam of radiation the daily tolerance dose is exceeded in three seconds.[5] He cannot possibly reduce a fracture in three seconds, and his only alternative is to use lead-lined gloves, a handicap with which it is almost impossible to manipulate fractures successfully. I have never met a surgeon practising the manipulative reduction of fractures under fluoroscopic screens who will swear that he never removes his gloves at a critical stage of the manipulation. If gloves are removed for no more than a few seconds, and the surgeon treats no more than four or five fractures a week, he is doomed. He will learn his fate only when it is too late. He will regret the day that he permitted himself to use a dangerous practice—dangerous not only to himself but also to his team.

Even if the surgeon does not put his hands in the direct beam, and uses no more than intermittent fluoroscopy by which to check the result of his manipulations, the daily tolerance dose from scattered radiation is reached within ten minutes. I would prohibit every surgeon from using the fluoroscope in reducing fractures. Equally good control is available by the use of ordinary films, developed rapidly and examined within two or three

[1] Ghormley, R. K., and Fairchild, R. D. "Surgical Treatment of Roentgen Dermatitis." *Surg. St. Louis*, 1940, 7, 737.
[2] Rigby, R. A. C., and Mowat, W. J. "X-ray Dermatitis." *Brit. med. J.*, 1941, 2, 770.
[3] Leddy, E. T., and Rigos, F. J. *Amer. J. Roentgen.*, 1941, 45, 696.
[4] Jupe, M. H. *Brit. med. J.*, 1941, 2, 175.
[5] Assuming average conditions, 30 ma., 60 kv., 1 mm. aluminium filtration, and a cone giving a circular field 5 in. in diameter at 30 in. focus-screen distance, the daily tolerance dose of 0.2 *r* is reached in the direct beam in 2.3 sec. at 20 in. distance and in 5 sec. at 30 in. (F. H. Kemp, "Fluoroscopic Control in the Reduction of Fractures." *Brit. med. J.*, 1942, 1, 39.)

minutes Even with this procedure the surgeon may be exposed to scattered radiation Moreover, he is also committed to the occasional task of holding a joint or fracture in a position of strain while radiographs are taken, and although lead-lined gloves should be worn almost invariably, this protection is sometimes impossible (as, for example, in testing for non-union of a small bone like the metacarpal) Finally, the surgeon is committed to the use of

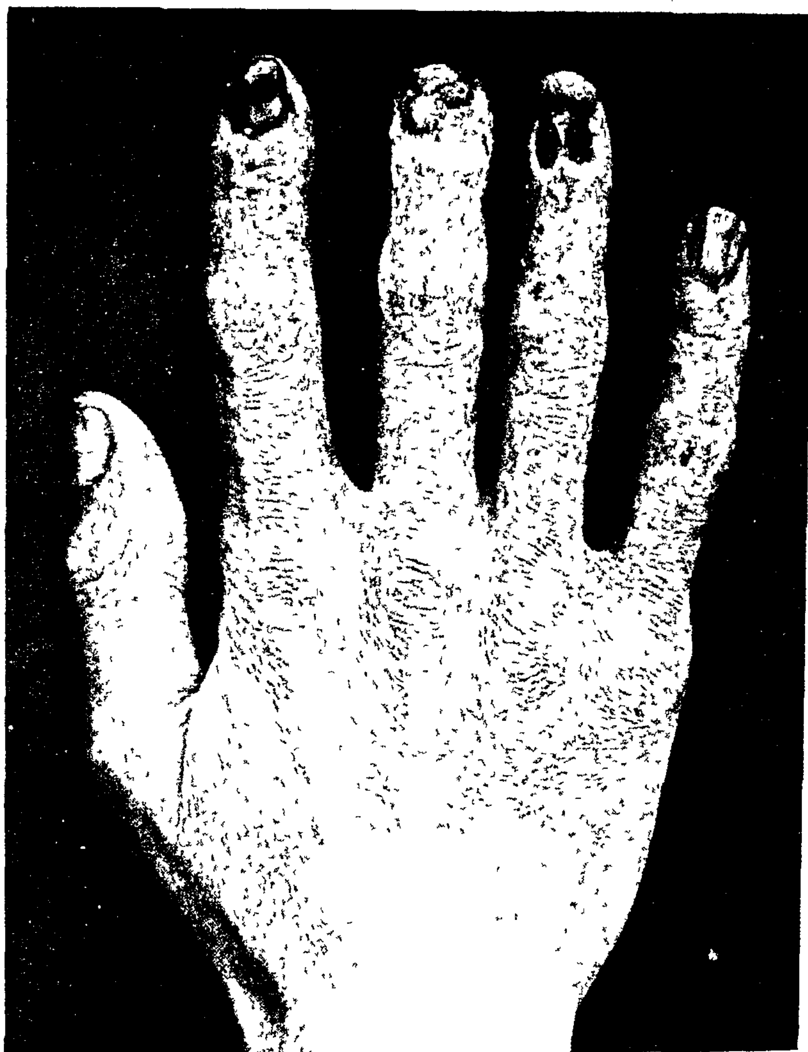


FIG 287
The result of exposure to X-rays Ninety-one surgeons are known to have paid this price (*By courtesy of the London Hospital and Dr Jupe*)

radiography during open operations when he cannot use lead gloves, this alone may be dangerous, and mechanical devices should be employed for holding the casette during nailing operations on fractures of the neck of the femur and *grafting operations on un-united fractures of the carpal scaphoid* bone. The very rare occasions when protection is impossible should be the limit of risk which the surgeon will accept Fluoroscopy must not be used The temptation is great, but the risk is greater Ninety-one surgeons are known to have paid the price. Don't be the ninety-second !

Dham

CHAPTER IX

MANIPULATIVE REDUCTION OF FRACTURES

Two of the three principles of fracture treatment, namely, those concerned with immobilisation of bone fragments, and with mobilisation of soft tissues and joints, were discussed in earlier chapters the third relates to reduction of the displacement These principles are equally important and each must be observed throughout all stages of treatment—

1. *Reduction*—Displacement of the fragments must be corrected and redisplacement prevented under radiographic control.
2. *Immobilisation*—The fragments must be immobilised completely and without interruption, until union is firm
3. *Functional activity*—Joints which need not be immobilised must be exercised actively but never stretched passively

Anatomically, the aim of treatment is to restore the bone to its former length, alignment and shape, physiologically, the aim is to restore joints and muscles to their former function Physiological recovery is accelerated by early functional activity which encourages repair of the bone, maintains a normal circulation, preserves the tone of muscles and the movement of joints, promotes mental recovery and prevents psychological complications. The ideal, therefore, is "early use of the injured part without movement of the injured structure" But this functional activity is safe only if redisplacement can be avoided Padded splints are inadequate, and closely fitting plaster casts, accurately moulded to the contour of the limb, are usually essential

METHODS OF REDUCTION AND IMMOBILISATION

The four methods of reducing and immobilising fractures are :

1 Manipulative reduction and plaster immobilisation
2 Manipulative reduction with continuous traction
3 Mechanical reduction and skeletal transfixion
4 Operative reduction and internal fixation

Manipulative reduction—Radiographs are inspected to see where the fragments lie The fragments are replaced by direct pressure or traction, and radiographic examination is then repeated in order to confirm that reduction has been secured If it is not complete, the routine of manipulation, fixation and X-ray examination should be repeated. Reduction must be achieved by guarded measured strength rather than sudden jerking force. In fractures near the ends of long bones considerable strength may be required ; it is difficult to over-reduce a Colles' fracture of the radius but easy to under-reduce it In many fractures of the ankle, and in epiphyseal separations, the fragments are so shaped that they lock when fully reduced, and it is impossible by manipulation to over-reduce them however strong the pressure that may be applied

In over-riding fractures of the shafts of long bones traction is necessary. It is a common mistake to apply perfunctory traction for only a few seconds Most fractured surfaces are irregular, and projecting spikes prevent the fragments from sliding into position. The limb must be slightly overlengthened before the serrations disengage, and slow steady traction must often be continued for several minutes.

Manipulation under anæsthesia—*To attempt the reduction of a fracture* without an anæsthetic is unfair to the patient and unfair to the surgeon Even the oldest patient can stand a few minutes of gas anæsthesia with safety. If, for constitutional reasons, it is believed that general anæsthesia is inadvisable, a local anæsthetic may be used

Local anæsthesia for fracture reduction—In this country, where general anæsthesia has reached a high standard of efficiency and safety, local anæsthesia is seldom used. The analgesia is not always perfect, and it is not suitable for children or nervous adults. On the other hand, it has the advantage of lasting for several hours so that manipulations may be repeated when necessary A long hypodermic needle is introduced through an intradermic wheal When blood can be withdrawn, confirming that the point of the needle is in the fracture-hæmatoma, from 10 to 20 c.c of 2 per cent novocaine are injected slowly. Analgesia should be complete within ten minutes If the fracture is impacted the novocaine must be injected subperiosteally at three or four points.

Brachial plexus anæsthesia—Regional anæsthesia is readily available in the upper limb by infiltration of the brachial plexus with novocaine. If the infiltration is successful there is complete analgesia lasting for several hours, thus permitting operations or repeated manipulations without discomfort or danger to the patient. The method was introduced by Patrick,[1] and is described in detail by Macintosh [2]

Standard of reduction—How perfectly must the fracture be reduced? The standard of end-result must be a limb clinically indistinguishable from normal, with normal function and normal appearance The alignment must always be perfect and there must be no rotational displacement Slight loss of apposition of the fragments may be of no significance in fractures of the shafts of long bones, especially in children. On the other hand, more severe lateral displacement causes bony thickening and impairs the cosmetic result, especially in a subcutaneous bone such as the tibia Even one or two millimetres of lateral displacement in a Colles' fracture of the radius gives an ugly deformity Moreover, lateral displacement may impair stability of the fragments Sometimes, therefore, anatomical reduction is necessary, but much latitude is often permissible in the apposition of fragments particularly in shaft fractures Angulation is not permissible even in the fractures of children

Time of reduction—Fractures may be reduced at any time during the first few days after injury Dislocations should be reduced at once because severe pain does not begin to subside until the displacement is corrected. With fractures there is less urgency Pain is relieved if the fragments are immobilised by first-aid splints, even if the displacement is not perfectly reduced

[1] Patrick, J *Brit J Surg*, 1940, 27, 734
[2] Macintosh, R R, and Mushin, W W "Local Anæsthesia Brachial Plexus" Oxford Blackwell Scientific Publications, 1944.

If the fracture is manipulated within an hour or two of injury, before there is reactionary swelling, great care must be taken not to apply so tight a plaster that the circulation might be endangered when the limb swells A plaster slab should be applied over half or two-thirds of the circumference of the limb and held in place with a soft bandage which may be cut if the necessity arises The encircling plaster is completed the next day If, on the other hand, twelve to twenty-four hours have elapsed and the limb is already swollen, even a closely applied plaster will become loose after a few days when the swelling subsides A new cast must therefore be applied after ten or fourteen days In some fractures of the ankle and leg, which have not been elevated or supported by firm elastic pressure, the limb may swell to almost twice the normal size In such cases the routine of delayed reduction may be preferable

Delayed reduction of fractures with severe swelling—Without preliminary manipulation and without anæsthesia, the fracture is immobilised by a plaster slab The patient is put to bed with the limb elevated. Two or three days later, when swelling has subsided, the fracture is manipulated and an unpadded plaster cast is then applied This routine is not applicable to dislocations, or to fractures where the displaced fragments may cause pressure on nerves or vessels. Delayed reduction of supracondylar fractures of the humerus is permissible only if there is no complication and if the circulation is undoubtedly normal Moore of Philadelphia has been so impressed with the frequency of early redisplacement that he invariably uses delayed reduction No attempt is made to reduce a closed fracture before the third or fourth day He finds that manipulation is facilitated and the incidence of secondary displacements reduced

Radiographic control of reduction—However satisfied the surgeon may be with his reduction of a fracture, there can be no excuse for neglecting to secure radiographic confirmation A post-reduction X-ray should always be taken It is true that the surgeon may decide to ignore certain displacements because he knows that the functional and cosmetic result will still be perfect, but unless radiographic examination has made him fully aware of the displacements that do exist, he is not in a position to know whether it is safe to ignore them I have seen several fracture-dislocations of the ankle, treated even by expert surgeons, where imperfect reduction was recognised so late that arthrodesis of the joint was the only available treatment.

If a limb is swollen at the time that the first plaster is applied, check radiographs should be taken ten days later, before the plaster is changed The fragments may have been redisplaced already Furthermore, the limb must again be X-rayed after applying the new plaster in order to confirm that the fragments still lie in the correct position Every time that a plaster is renewed this routine should be practised, no matter how unlikely it may seem that the fragments have moved

Certain fractures are prone to redisplacement within the plaster even despite satisfactory initial reduction and a closely fitting plaster cast. This applies particularly to fractures of the lower shaft of the radius with inferior radio-ulnar dislocation, fractures of both forearm bones, fracture-dislocations of the ankle, and fractures of the shaft of the tibia These fractures should be X-rayed every second or third week during the first two months

PLASTER-OF-PARIS TECHNIQUE

Many thousand years ago the Egyptians immobilised fractures by linen stiffened with gum or plaster. Starch, clay and egg albumin were employed. In the last century walking plasters were used by Krause in the treatment of leg fractures and, in 1887, he reported a successful series of ninety-eight cases. He fitted a special laced boot over the plaster for

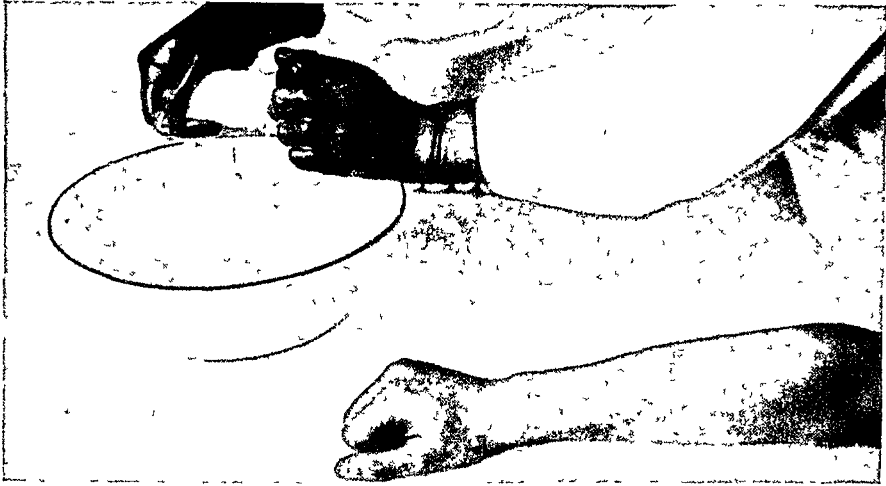


FIG. 288

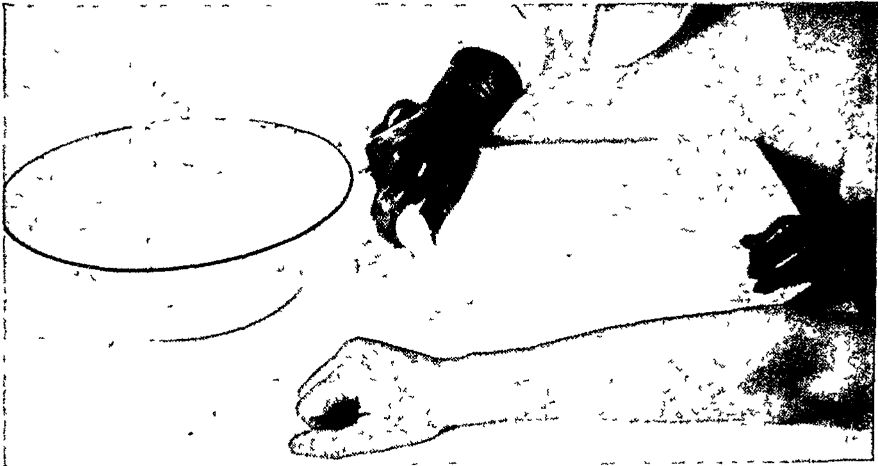


FIG. 289

Plaster slab technique. The bandage is soaked until bubbles cease to rise. While holding each end it is lightly squeezed. The wet slab is rapidly prepared.

walking out of doors. Unpadded plasters and U-shaped walking iron stirrups were employed by Korsch in 1894.[1] More recently the unpadded plaster has been popularised by Bohler and many other surgeons.

Preparation of plaster bandages—The best dental plaster should be used and a fairly wide-mesh starch-free crinoline or muslin bandage, six inches wide and five yards long. The plaster is firmly and evenly rubbed into the crinoline, which is lightly rolled. If the rolling is too loose, the central core slides out during application; if it is too tight, the bandage will not

[1] Monro, J. K. "History of Plaster-of-Paris in the Treatment of Fractures." *Brit. J. Surg.*, 1935, 23, 257.

soak evenly The completed plaster bandage should be elastic and springy. The proportion of plaster to crinoline depends on individual preference but too much crinoline makes an expensive bandage and too much plaster a brittle cast An average five-yard plaster bandage should weigh eight ounces and contain 85-90 per cent. by weight of plaster

Plaster slab technique—The plaster bandage is lowered into a deep bowl of warm water and left completely immersed until bubbles cease to rise.



FIG. 290

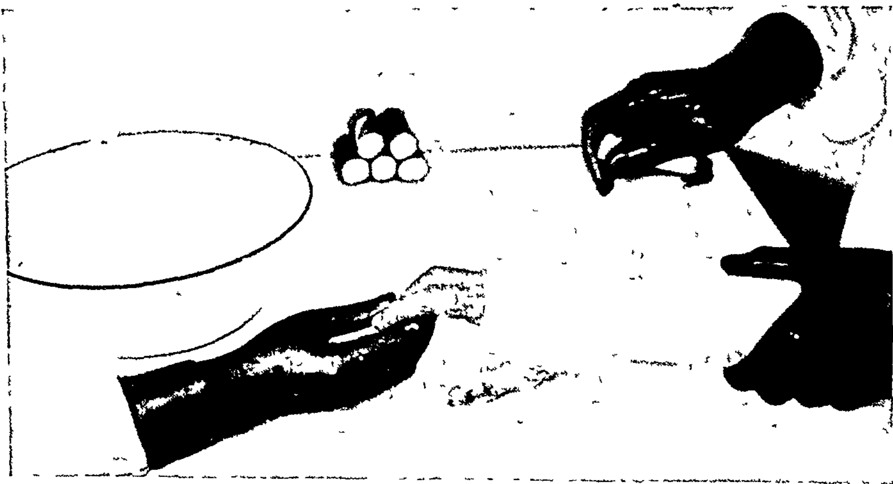


FIG 291

Plaster slab technique. The plaster is moulded to the contour of the limb and held in position with wet open-wove bandage

It is then squeezed lightly from the two ends A slab of the required length and width is prepared on a glass or enamelled surface (Figs 288-291). A proper cast is not a series of discrete layers of plaster but one homogeneous mass. The bandage must therefore be sufficiently wet and sloppy , each layer must be firmly rubbed in , and the work must be completed rapidly. While the plaster is still wet, and before it has had sufficient time to set, it is applied to the limb, firmly moulded round the bony prominences and bound lightly in position with a *wet* open-mesh bandage The encircling

bandage must not be dry because, after application, as it becomes moist from the wet plaster, it shrinks exactly as the unloosened guy ropes of a tent shorten and snap in rain Movement of the joints during setting of the plaster produces ridges at the joint level, sometimes with disastrous results. This must be avoided by keeping the limb immobile until the plaster is hard

To immobilise a wrist joint, the slab is made of sufficient length to allow it to be turned back double over the hand and wrist. Similarly, an elbow joint may be immobilised by a plaster slab down the back of the limb For an ankle joint, more than one slab may be used one is applied from the upper calf, over the heel and sole to the toes ; and a second longer slab is applied down one side of the limb, under the heel, and up the other side.

Complete plaster cast—A complete cast is constructed by applying one or more slabs, carefully moulding them in position, and then applying ordinary plaster bandages in a circular manner. *These encircling turns of plaster bandage must not be pulled tightly ; they should be laid on the limb without tension* When necessary, a slight pleat is taken in one margin of the bandage in order to preserve an even and smooth application Each layer is firmly rubbed in and the plaster, while still wet, is moulded round bony prominences to conform exactly to the contour of the limb This moulding is done with the palm and thenar eminence rather than with the finger-tips

Padded or unpadded plasters ?—When a limb is put in plaster shortly after injury, and before it has begun to swell, padding should always be used A double-faced wool bandage can be applied smoothly and evenly Similarly, after operations performed with a tourniquet, operative excision of wounds or compound fractures, sequestrectomy, or any other operation that may cause reactionary swelling, the plaster should be padded On the other hand, when the plaster is changed after two or three weeks, and it is known that there will be no further swelling, it is better to use an unpadded cast The plaster must fit very closely . it may be applied directly to the skin or over a single layer of stockinet Bony prominences, particularly the neck of the fibula, the iliac spines, and the spinous processes, should be protected with ¼-inch adhesive felt Friction at the upper and lower margins of the cast should be prevented by a strip of felt or wool.

Splitting the plaster—Whether it is padded or not, a complete plaster cast that is applied within a few hours of injury, or immediately after operation, should be split throughout its length The limb must be elevated until the circulation is re-established and the swelling and œdema controlled

A complete plaster cast must not be used if the circulation is in doubt—When vascular damage is suspected, encircling plaster is not permissible until the danger of circulatory complication has passed. Meanwhile, skeletal traction should be relied upon, or if necessary a simple plaster slab may be bandaged lightly to the limb.

Wedging of plasters—It is of great value to be able to correct minor degrees of angulation without completely changing the plaster If the plaster is cut down, and a new one applied, some other displacement may arise and the surgeon is tempted to "leave well alone" Yet the slightest degree of angulation should be corrected and a simple device is available by which this can be done with perfect control

A linear cut is made round two-thirds of the plaster at the level of fracture on the concave side of the angle.[1] A short anæsthetic may be given, and the linear division is opened to a wedge which is held open by a small block of wood placed between the two cut edges (Figs 292-294). The block must be exactly opposite the angle, in front of the limb for backward angulation, on the inner side for outward angulation, half-way between the two for combined backward and outward angulation, and so on Another radiograph is then taken and the degree of correction may be

FIG 292

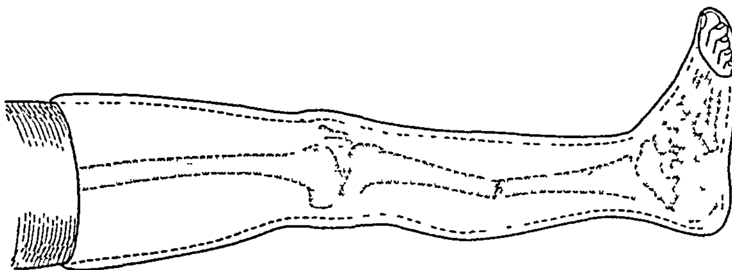


FIG 293

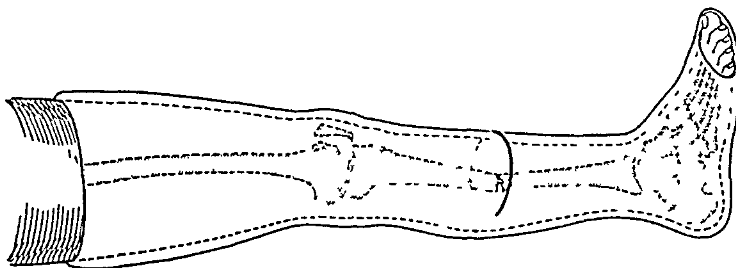
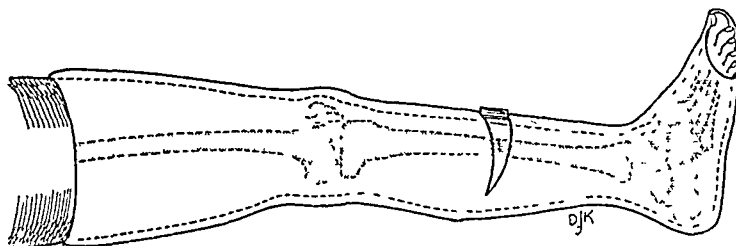


FIG 294



Wedging a plaster to correct angulation

A linear cut is opened to a wedge, held by a wood block, and after confirming the position by X-ray, the gap is repaired with plaster

(*By courtesy of " J Bone Joint Surg ," from author's article*, 1932, **14**, 591)

increased or decreased by inserting larger or smaller blocks of wood When a final radiograph proves that alignment is perfect, the gap in the plaster is filled and reinforced

This procedure is invaluable in the treatment of fractures of the tibia and fibula It allows perfect control and accurate correction of the slight degrees of angulation which cannot always be avoided at the time of the first manipulation. It may also be used in fractures of the shafts of the

[1] Instead of adopting the simple plan of making a linear cut in the plaster on the concave aspect of the angulated bone and opening the cut to a wedge-shaped space which is filled in, the opposite procedure may be practised of removing a wedge of plaster from the convex aspect and closing the gap This was described by Pierre Godenne in 1920 (*J Chir , Brux* , March 1920) Alternatively the still more complicated, though theoretically perfect, device may be adopted of cutting the plaster on both concave and convex aspects, leaving rather less than 1 inch of plaster intact on each side, and thereafter opening one cut and closing the other These alternatives have been suggested in order to avoid any possible danger of distraction of the fragments when using the method described in the text—but I think that the danger is largely theoretical

forearm bones, and in trochanteric fractures of the femur treated in a hip spica (Figs 295-296) Caution is necessary if it is proposed to correct severe degrees of angulation; such wedging may so increase the pressure of the plaster on the limb, even at a distance above and below the level of the wedge, that pressure sores may develop

Walking plasters—A walking plaster for the lower limb should be reinforced with an extra slab beneath the sole extending to just above the back of the ankle. Irregularities are made good and a flat walking surface produced The patient should not walk until the plaster is thoroughly dry The impact of weight-bearing may be reduced by applying a ¼- or ½-inch heel of sorbo rubber held in position by a few turns of adhesive





Fig 295 Fig 296

Wedging a hip spica to increase the abduction of a trochanteric fracture Radiographic control is used

strapping A plaster boot is made with a leather sole so that the patient can walk out of doors even in wet weather and appear normally dressed (Figs 297-298)

Walking iron stirrups projecting beyond the heel are often incorporated in the plaster Although they reduce wearing of the plaster most patients acquire a bad gait Either they walk rigidly as if on a stilt, with the knee stiff, or they pivot on the heel, twisting the whole limb out with each step. It is very much easier to achieve a normal heel and toe movement with an ordinary plaster, with or without a sorbo rubber heel Moreover, it is difficult to fit a boot over a walking iron stirrup, and a closely fitting plaster boot adds much to the patient's self-respect, it enables him to walk in streets and shops almost unnoticed Many patients have learned to pursue a normal life despite a below-knee walking plaster, cycling, driving, riding, playing golf, and even hunting and deer-stalking!

Removal of plasters—A professor of surgery retains one vivid childhood memory, namely, the agony of removal of a plaster. The pain of the fracture is forgotten but not the pain of cutting the plaster. Even the most gentle surgeons seem to lose their sympathy on these occasions; they brush aside the victim's protests and hasten through the work as if time was the only factor. One patient's thigh was completely split open from top to bottom by the cutting edge of a plaster shears wielded by a vigorous young house surgeon. The patient was under an anæsthetic, but forty stitches in a fourteen-inch wound were not easily explained.

The first essential is not to frighten the patient, and to realise that even the sight of plaster shears may terrify a child. The second is to choose



Fig. 297



Fig. 298

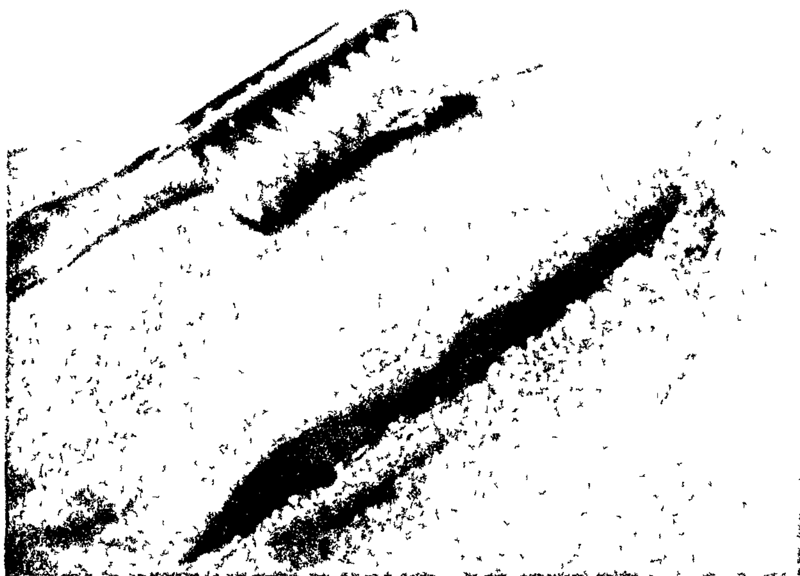
Walking plaster. A sorbo heel is incorporated, a plaster boot is worn and the patient dresses normally and pursues normal activities.

good shears. The blade that is inserted beneath the plaster should be slightly longer than the other, so that it does not cut out with each bite; but it should not be more than $\frac{1}{8}$ inch longer and it must be shallow. Much of the pain and discomfort is due to the surgeon's struggles to push the deep blade in advance of the cut. The third precaution is to use the shears gently. The deep blade must be kept parallel with the skin and not pointed down at it. Short bites should be taken. 4 Special care is necessary at joint levels where bones are prominent beneath the skin. Over these areas the plaster should be nibbled away a millimetre at a time.

Motor saws have been devised for cutting plasters but they have proved unsatisfactory. They are difficult to use and they frighten the patient. The safest is the Stryker saw which has an oscillating, but not a revolving, blade so that the danger of cutting the skin is minimised. It still makes a terrifying noise; and even if it is harmless it looks and sounds as if it will hurt.

Protection from disuse œdema—As soon as a lower limb plaster has been removed an elastic support must be applied. The circulation has become accustomed to rigid external support and when this is lost suddenly, pronounced œdema may develop. If œdema is left uncontrolled it may recur every day for many months or even years. Every time that the fluid leaks from the capillary field into the tissue spaces it opens tracks which make it easier to leak again. The œdema causes adhesions, and the weight of the swollen limb interferes with the exercise that is necessary to promote venous return and restore the circulation. Massage is of little benefit; it only disperses the fluid temporarily.

There should be no interval between removal of a plaster and the application of an elastic support. If, in error, an interval has been allowed and the limb is already swollen, the patient should be put to bed for twenty-four hours with the foot elevated before the dressing is applied



A child whose fractured tibia was immobilised in a plaster cast told his mother that he had lost a tooth brush under the plaster She refused to believe him Three weeks later, after he had been asked to leave school because "he smelled," removal of the plaster revealed the tooth brush and the pressure sore it had caused

Fig 299

Ordinary crepe bandages are not altogether reliable because they tend to slip; and if the patient applies the upper turns of the bandage more tightly than the lower, the œdema is aggravated An elastic stocking is more effective but it is expensive and liable to stretch Elastic adhesive strapping often causes skin irritation and dermatitis

Unna's zinc gelatine paste is the most useful dressing. The cubes of gelatine are placed in a pot surrounded by water which is boiled until the gelatine melts to a smooth paste It is then painted on the leg and covered with a soft encircling bandage Two or three alternate layers of paste and bandage complete the dressing which should extend from the webs of the toes to just below the knee Proprietary bandages already impregnated with a gelatine paste may be used "Viscopaste" bandage requires no preparation but dries with a slightly harsh surface "Ichthopaste" bandage requires immersion in hot water for ten or fifteen minutes and at first smells of ichthyol, but it is non-irritant and soothing to a tender skin.

The dressing should be retained until the muscle tone and circulation of the limb are restored The patient's observation that the dressing is no tighter at the end of the day than it was at the beginning shows that the tendency to œdema has subsided. In the adult, protection is necessary for five or six weeks and sometimes for several months.

COMPLICATIONS OF PLASTER IMMOBILISATION

Pressure sores may develop from the localised pressure of splints, bandage or plaster, especially over bony prominences. Plaster sores may arise from pulling one turn of the plaster bandage so tight that there is a ridge on the deep surface, careless moulding of the plaster, causing undue pressure over a bony prominence, application of a plaster slab which instead of being wet and sloppy has begun to harden and does not adapt itself smoothly to the contour of the limb, movement of a joint during the setting of a plaster so that a ridge is formed, failure to protect bony prominences in a thin or emaciated patient, allowing an imperfectly set plaster to rest on a hard surface so that it is flattened over a bony prominence (particularly the back of the heel in leg plasters and the sacrum in hip and trunk plasters), pushing coins, heads of knitting needles, small wads of wool or other foreign bodies between the plaster and the limb (Fig 299), or delay in repairing a crack in the plaster near a joint so that there is friction of the skin against the broken margins



Fig. 300

A plaster sore

The patient's complaints of minor pain and discomfort were ignored because he was a querulous "difficult" type who was always complaining Even minor complaints must always be accepted, a window must be cut or the plaster bivalved

Signs—The patient may complain of persistent localised discomfort or pain These symptoms should never be ignored, even if they pass off in a day or two, because the tissues soon become anæsthetic. Sometimes there has never been any complaint of pain and the first sign may be the smell of accumulated secretions and discharge The sore can easily be localised because the overlying plaster becomes much hotter than elsewhere Oedema of the toes and fingers, recurring after the initial œdema has subsided, means almost certainly that there is a pressure sore If the sore is near the end of the plaster the digits become red and inflamed, or dusky Finally, sloughing is recognised by staining of the overlying plaster or by purulent discharge from the end of the plaster

Treatment—As soon as a pressure sore is suspected a small window must be cut (Fig. 300) Even if the suspicions were unfounded no harm has been done and the patient must not be allowed to feel that he has complained unnecessarily. Whether or not there is a sore, the window must be filled with a pad of wool bandaged firmly into position. Unless this is done

there will be œdema of the unprotected area which aggravates threatening ulceration, or even produces new sores round the margin of the window. If there is already ulceration a moist dressing should be applied until sloughs have separated, and then a dry or vaseline gauze dressing which is not changed more than once a week If a large area of skin has been destroyed much time may be saved, and functional incapacity prevented, by the early application of a split skin graft, or by mobilising and transposing whole-thickness flaps of skin

Oedema distal to the plaster—Oedema of the fingers and toes is inevitable after injuries of the wrist and ankle, particularly when local swelling is prevented by a plaster cast. This reactionary swelling is of little significance but the limb should be elevated until it has subsided Active exercise of the swollen digits is of the greatest importance It is a mistake to try to prevent swelling of the toes by cutting the lower margin of the plaster back to a more proximal level In the lower limb the œdema is gravitational, and the more the plaster is removed the greater is the area that will swell, and the more inevitable are pressure, friction and ulceration of skin at the margin of the plaster (Fig 301) For the same reason a plaster spica, or a plaster for the knee, should never extend only to a level above the ankle joint, thus leaving the foot unprotected If the ankle is not to be immobilised a viscopaste bandage must be applied to the foot from the webs of the toes, incorporated in the lower margin of the plaster



Fig 301

Paste dressing, crepe bandages and plasters must extend to web of toes, otherwise blistering and gravitational œdema develop in the unprotected forefoot, and there may be ulceration at the margin of the dressing

Purulent dermatitis—When plaster is applied directly to a limb without padding, the skin usually becomes dry and scaly Some patients have a susceptible skin and dermatitis develops, similar to intertrigo dermatitis Staphylococcal infection of the hair follicles and sweat glands supervenes and if the condition is ignored severe purulent dermatitis may arise The first sign is itching and irritation, followed later by intolerable burning pain At the first sign of irritation a small window should be cut, talcum powder should be blown under the margins of the plaster every day. It is sometimes necessary to apply a new plaster over a double layer of stockinet

Skin blistering is common during the first twenty-four hours after severe injuries of the ankle, leg and elbow The blisters are due to traumatic œdema of the cuticle, and the exudate is sometimes hæmorrhagic They develop only where the skin is unsupported If splints are used they appear between the splints, not beneath them They never develop beneath an encircling plaster If the skin is already blistered the blebs should be emptied by pinching a minute hole in the overlying cuticle Penicillin-sulpha powder is dusted over them and plaster applied in the usual way.

5 **Gangrene**—Gangrene arising after a fracture is usually due to arterial injury and it may be an unavoidable complication In recent years there have been many tragic cases of gangrene where the pressure of an unpadded plaster was the most important if not the only cause The circulation must be tested frequently It is not enough to compress the tips of the fingers or toes and confirm that the anæmic area refills with blood , fluid blood often remains in the digit, and this sign may be observed even after complete stasis of the circulation The anæmic area should flush rapidly with blood. The digits should be warm , they should not be cyanosed or pale , they should be pink If return of blood after compression of a digit is slow the patient must not be allowed out of sight The test should be repeated every ten or fifteen minutes If the digit is blue, or cold and pallid, the plaster must be cut at once The limb should be elevated in order to assist venous drainage and reduce the œdema It is usually sufficient to make a longitudinal cut through the whole length of the plaster, dividing every turn of wool or bandage until the skin is exposed in the gap between the cut edges The gap should then be filled with a light padding of wool If necessary the whole of the front half of the plaster should be removed. If the pallor and anæmia are still unrelieved, it is clear that the artery has been injured or that it is thrombosed

It must be emphasised that it is not enough simply to cut the plaster. Very often it is not the plaster that is tight but the bandage or wool beneath it In a recent disastrous case, in which I operated on an un-united fracture of both bones of the forearm, there was circulatory embarrassment some hours later and the plaster was promptly cut throughout its length—but most unfortunately the skin was not exposed in the cut and it was not until the next day that a further cut was made in the underlying wool, which was impregnated with blood and, by then, was board-like, almost concrete-like, in its hardness It was the petrified wool that was responsible—and it was cut too late Gangrene was avoided but the limb was crippled by ischæmic contracture *At the first sign of circulatory embarrassment the plaster, and every layer of bandage or wool beneath the plaster, must be cut until skin is exposed widely in the gap*

Gangrene complicating fractures with burns — The circulatory embarrassment that leads to gangrene is the consequence of reactionary swelling and œdema continuing after the application of a rigid plaster cast The danger is greatest when a fracture is associated with a major burn of the same limb , to the traumatic œdema and hæmorrhage associated with the fracture is added the inflammatory œdema and exudation associated with the burn There may be tremendous swelling which increases steadily during the first thirty-six hours If an unpadded plaster cast, or even a padded cast, is applied on the first day of injury, gangrene is almost inevitable [1] This association of injuries is not uncommon in aeroplane crash landings, where the wreckage is so often consumed in flames Every such case dealt with in the Royal Air Force orthopædic service during the recent war was complicated by gangrene and amputation of the limb when plaster was used during the first twenty-four hours (Figs 302-305).

Even apart from the immediate danger of ischæmia due to pressure of the plaster, there is a later danger of lymphatic spread of infection from

[1] McIndoe, A , and Watson-Jones, R " Unpadded Plaster, Rigid Dressings and Gangrene " *Lancet*, 1944, 1, 738

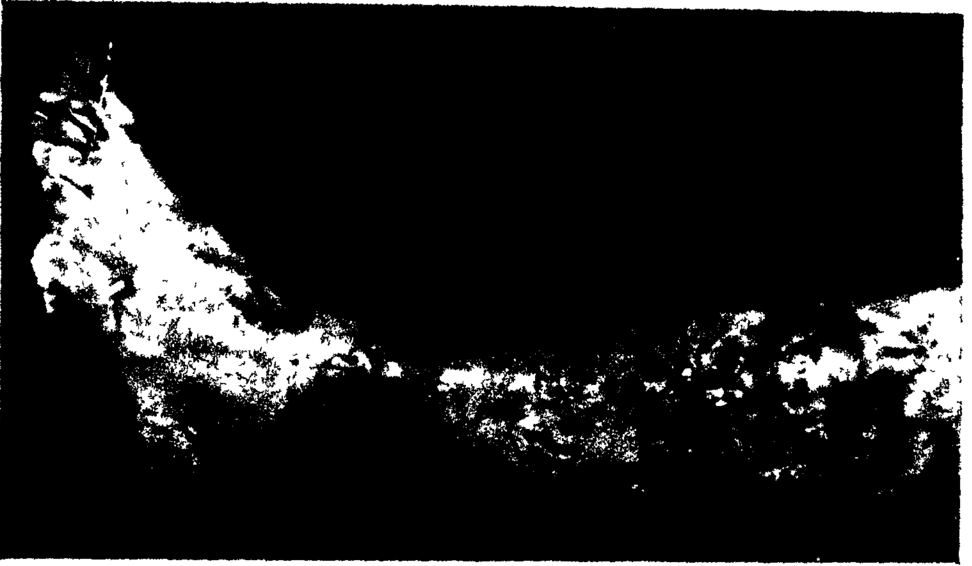


Fig. 302



Fig 303

Figs. 302-305 show the lower limbs of a pilot who sustained bilateral fracture-dislocations of the ankle joints with bilateral third degree burns.

the burnt skin and subcutaneous tissues to the undrained hæmatoma of the fracture. The problem of treatment may thus be increased very greatly by infection of the fracture and the bone No matter how great the difficulties of immobilising the limb, a complete plaster cast should *not* be used The first objectives are the treatment of the burn, the control of infection and the replacement of skin, and in achieving these ends it matters little that deformity may arise, it is easy to correct deformity by later reconstructive operations, but it is not possible to replace a limb that has been amputated



Fig 304

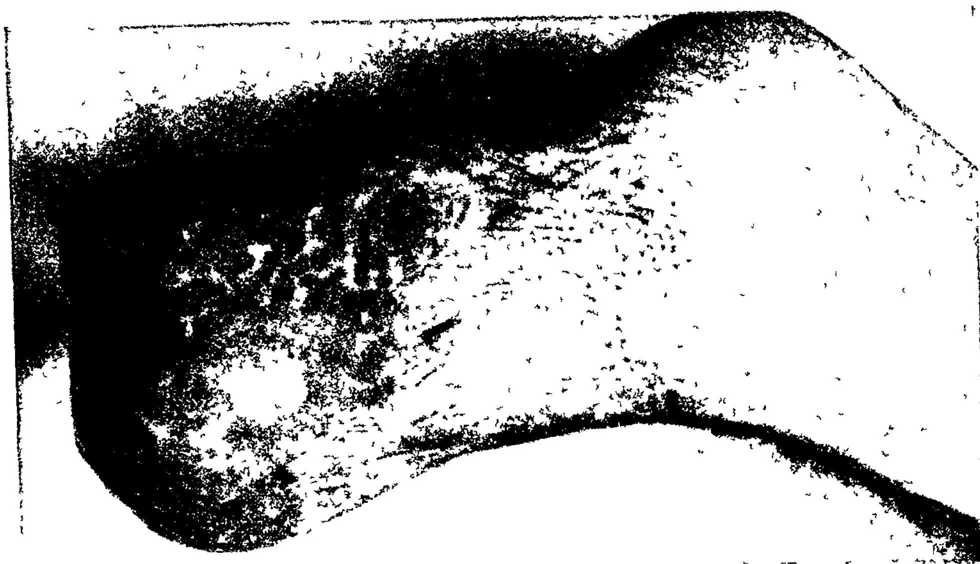


Fig 305

Figs. 302-305—Unfortunately the combined fractures and burns were treated by first-aid application of unpadded plaster casts Gangrene and amputation was the inevitable consequence

GENERAL COMPLICATIONS

Hypostatic pneumonia—When an elderly patient sustains an injury necessitating recumbency there may be a tendency to develop hypostatic congestion of the lungs. leading sometimes to pneumonia This is increased if the patient is unable to move because he is immobilised in a heavy plaster cast. Regular turning from one side to the other, and when possible on to

the face, should be carried out every few hours As soon as possible the patient should be propped up with pillows

Renal calculus formation—If recumbency is necessary for several months it should be borne in mind that there will be a generalised disuse decalcification of the skeleton with a greatly increased output of calcium salts by the kidneys To the generalised decalcification of all bones may be added the more marked local decalcification of the fractured bone, particularly when



FIG 306

Nephrolithiasis due to recumbency in a case of fracture-dislocation at the eleventh and twelfth dorsal level

the fracture is infected High concentration of calcium salts in the urine may be responsible for extensive calculus formation (*Fig 306*) At first the calculi are soluble and they often disappear when the saturation of calcium salts in the urine is reduced by increased fluid intake and drainage of the renal tract is improved by posture Meanwhile, however, there is a risk of renal insufficiency from obstruction, and the excessive excretion of calcium itself leads to renal cellular degeneration Moreover, deposition of calcium salts may provide a nucleus for the formation of insoluble calculi In some cases pyonephrosis has led to loss of a kidney or even death from uræmia

Especial caution is necessary in compound fractures of the lower limb, in fractures of the hip, in injuries of the pelvis with rupture of the bladder or urethra and in spinal injuries treated in recumbency Postural drainage of the kidneys should be encouraged by raising the head-end of the bed on blocks for several hours each day The fluid intake should be increased and regular exercises practised for those parts that do not need to be immobilised In order to make the urine acid and so maintain solution of the urinary calcium phosphate, a diet yielding an acid "ash" is recommended Details of an acidogenic diet are given by Pyrah and Fowweather [1]

3 **Fat embolism**—Of the three complications of fractures that may cause sudden death, shock is usually fatal at about the third hour, fat embolism on the third day, and pulmonary embolism during the third week Fat embolism, though seldom recognised clinically, is the cause of death in over 20 per cent of fatal fracture cases [2, 3] It has been assumed that the fat globules gain entrance to the circulation from the medulla of the injured bone through the ruptured walls of blood vessels, but it is also possible that the emboli may consist of the fat of blood plasma, broken down from its normal state of emulsification by histamine or other products of muscle injury, or by ether inhaled during anæsthesia [4, 5] The emboli lodge in the lungs or brains, or are scattered throughout the body. The pulmonary type sometimes develops within a few hours of injury and clinically it resembles acute œdema of the lungs, but more often dyspnœa, cyanosis, precordial pain, cough and hyperpyrexia develop gradually during the first few days and simulate broncho-pneumonia In the cerebral type, the symptoms are those of delirium tremens, and cerebral irritation passes into stupor, coma and death

Diagnosis—Although the diagnosis of fat embolism is seldom established before death of the patient, it can be made if certain physical signs are deliberately sought [6] Mental disturbance, and alternation of coma with full consciousness, occurring some hours after a major bone injury, should put the surgeon on guard He should examine the neck and upper trunk for petechial hæmorrhages He should turn down the lower lid of the eye to seek petechiæ He should examine the fundus and, if it is indeed a case of fat embolism, he will nearly always find yellow-white glistening patches of perivascular œdema, areas of perivascular hæmorrhage, or even globules of fat. Very occasionally there is fat in the sputum [7] or fat in the urine, but these signs are unreliable

Treatment—The only recognised treatment is that of prevention, the gentle handling of fractures in first-aid and subsequent treatment, the institution of complete immobility at the first possible moment, and the use of tourniquets for all bone and fracture operations [8] In fractures of the femur the possible benefit of early ligation of the profunda femoris vein has been considered by Newman [6]

[1] Pyrah L N, and Fowweather, F S "Urinary Calculi in Recumbent Patients" *Brit J Surg*, 1938, **26**, 98
[2] Rowlands, R A, and Wakeley, C P G "Fat Embolism" (full bibliography) *Lancet*, 1941, **1**, 502
[3] Vance, B M *Arch Surg*, 1931, **23**, 424, and *Amer J Surg*, 1934, **26**, 27
[4] Watson "Fat Embolism" *Brit J Surg*, 1936-37, **24**, 676
[5] Lehmann and Moore *Arch Surg*, 1927, **14**, 621
[6] Newman, P H "Clinical Diagnosis of Fat Embolism" *J Bone Joint Surg*, 1948, **30-B**, 290
[7] Scott, J C, Kemp, F H, and Robb-Smith, A. "Fat Embolism Sputum Examination" *Lancet*, 1942, **1**, 228
[8] Ryerson, E W *J Amer med Ass*, 1916, **67**, 657

MANIPULATIVE REDUCTION AND CONTINUOUS TRACTION

"*Two strong men will suffice by making extension and counter-extension*" —HIPPOCRATES, 350 B.C

Principles of traction—When the shaft of a long bone is fractured the elastic retraction of muscles surrounding the bone tends to produce over-riding of the fragments. This tendency is greater when the muscles are powerful and long-bellied as in the thigh, when the fracture is imperfectly immobilised so that there is pain and therefore muscle spasm, and when the fracture is mechanically unstable because the fragments are not in apposition or because the fracture line is oblique.

Upper limb—In injuries of the upper limb, the first and second aggravating factors can be excluded, the muscles are not especially strong or long-bellied, and there is no difficulty in securing complete

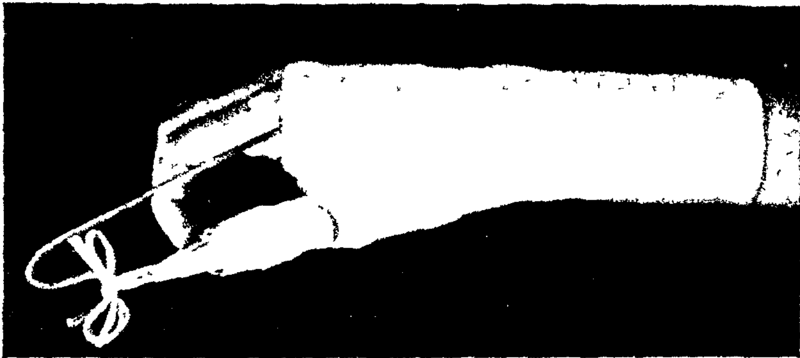


FIG. 307
Traction in the upper limb is seldom required. Even thumb traction for Bennett's fracture-dislocation has disadvantages and may well be unnecessary if the plaster is moulded carefully enough

immobility and therefore in relieving muscle spasm. Even in Bennett's fracture-dislocation of the thumb metacarpal, if the plaster is moulded carefully it may be quite unnecessary to use continuous traction, which has definite disadvantages in causing stiffness of the joints. Traction may occasionally be advisable in unstable fractures of the neck of the humerus when the limb must be immobilised in abduction and yet the shaft tends to slip below the proximal fragment; but again there are disadvantages in traction and the alternative of open reduction with internal fixation by an intramedullary pin must be considered.

Lower limb—In the lower limb, the power of muscles and the range of retraction is greater and most fractures of the shaft of the femur require traction throughout the period of immobilisation. Open and infected fractures of the leg bones sometimes need traction for a few weeks. Whenever possible, however, continuous traction should be avoided in fractures of the shaft of the tibia because there can be no doubt that it slows the rate of union. For unstable fractures of the tibial shaft it is usually better to use internal fixation.

Principles of counter-traction—A team practising tug-of-war might possibly fix their rope to a wall but usually they pull against another equally strong team. Similarly, traction on a limb demands either a fixed point

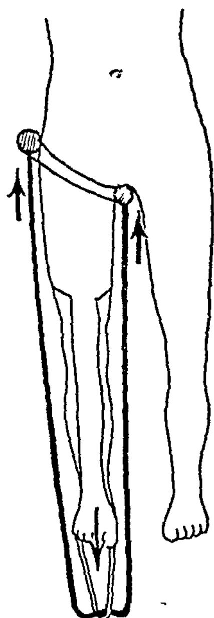


Fig. 308

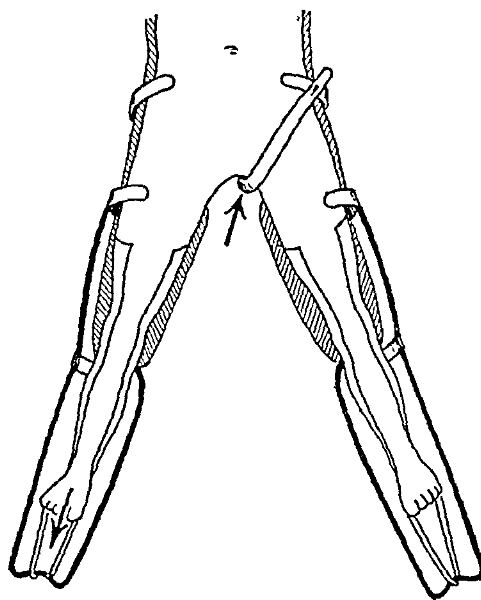


Fig 309

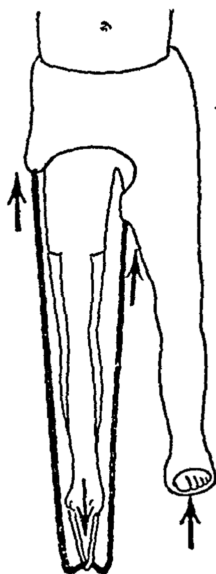


Fig 310

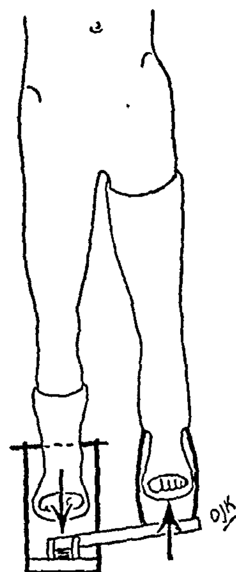


Fig 311

Fixed types of lower limb traction The counter-pressure is against the pelvis in the Thomas' splint (Fig 308), the opposite groin in the abduction frame (Fig 309) and the sole of the opposite foot in "well-leg" traction plasters and splints It should be noted that "well-leg" traction is now almost obsolete (Figs 310-311)

from which the traction may be exerted, or an equal counter-traction in the opposite direction Each of these methods is employed The first is known as fixed traction and the second as balanced traction.

Fixed traction—With a Thomas' bed splint, traction is exerted from the fixed point of the patient's pelvis The extension tapes pull the limb down to the splint which is prevented from moving in the opposite direction by the resistance of the ring of the splint against the tuber ischii (Fig 308). With an abduction frame, traction is exerted from the fixed point of the

groin strap on the opposite side (Fig 309) "Well-leg" traction is exerted from the fixed point of the sole of the opposite foot[1] This method was devised in order to avoid the pressure of the ring of a Thomas' splint in the groin The opposite limb is put in a plaster spica and the bars of the splint are incorporated in it (Fig 310) The counter-pressure is then transferred from the groin to the plaster spica and therefore to the sole of the opposite foot This principle is used in the Roger Anderson splint for certain fractures of the upper end of the femur. Although with this splint the plaster on the normal limb is not a complete spica, the counter-pressure is transferred to the opposite sole in a similar way (Fig. 311)

Balanced traction—The simplest type of balanced traction is that used in weight-traction. A weight is hung over a pulley at the foot of the bed and fixed to the limb by strapping on the skin, or by a traction pin through the bone The counter-traction is the patient's body-weight sliding down the bed which is raised at the foot (Fig 312). The 10 or 15 lb weight on one side of the pulley is balanced by the patient's own weight on the other This principle is used in Braun's splint for fractures of the femur and tibia, and in Hodgen s splint or any other splint which is suspended from an overhead beam

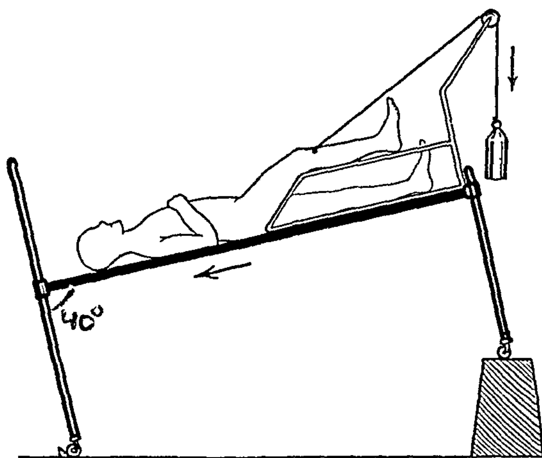


Fig. 312

Balanced traction The traction of the weight on one side of the pulley is balanced by the counter-traction of body-weight on the other

Combined fixed and balanced traction—Most surgeons who use the Thomas' bed splint regularly also use combined fixed and balanced traction The splint is applied in the ordinary way with the ring firmly pressed against the pelvis and the traction tapes tightened As the traction continues the tapes tend to slide and slacken so that it is necessary to tighten them several times a day. Furthermore, pressure of the ring of the splint causes discomfort, or even pressure sores, in the adductor region and groin Both these difficulties are met by fastening the end of the splint to the foot of the bed, which is raised about eighteen inches, the patient is then partly suspended from the foot of the bed by the tapes, some degree of balanced traction has been added, the patient tends to fall away from the ring of the splint and pressure in the groin is reduced, and moreover, slack in the traction tapes is at once taken up so that they need tightening only once a day or on alternate days Similarly, with an abduction frame the efficacy of traction can be increased, and the discomfort of the groin strap relieved, by fastening the frame to the raised foot of the bed

Sliding bed traction—Pugh first utilised the simple device of applying traction tapes to the limb and fastening them to the raised foot of the bed If only one limb is fastened in this way the opposite side of the pelvis slides

[1] Anderson, Roger "A New Method of Treating Fractures utilising the Well Leg for Counter-traction" *Surg Gynec Obstet*, 1932, 54, 207

down the bed more than the fixed side, and the hip is slowly abducted. The device is useful in treating epiphyseal coxa vara and avascular necrosis of the upper femoral epiphysis, where traction and abduction are required, but not immobilisation The extent to which the patient slides down is reduced by friction against the mattress and bed clothes. Hendry has excluded this friction in a "sliding bed." The mattress is placed on a wooden surface on roller bearings (Fig 313). The amount of traction that demands a tilt of 30° or 40° on an ordinary bed can be achieved on a sliding bed with only a 10° or 15° tilt The available traction is almost unlimited and the comfort of the patient is greatly increased. Actually the head of the patient need not be any lower than his feet, not even 10° or 15°. Provided that the bed itself is raised at the foot, the false top will still slide down and give the necessary traction whether it is parallel with the bed or not; and in the latest model of Hendry bed the sliding top is horizontal, although the bed on which it slides is tilted

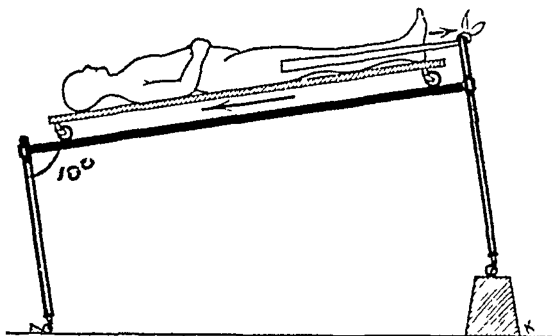


Fig 313

Sliding bed traction (Hendry and Naughton Dunn) By excluding friction, a 10° tilt on a sliding bed gives as powerful traction as a 40° tilt on an ordinary bed

Dangers of traction and distraction—A clear distinction must be drawn between the heavy traction that may be applied for a few minutes as part of the manœuvre of manipulative reduction of a fracture, and the lighter traction that is maintained continuously in order to prevent redisplacement Continuous traction should not be used for the reduction of a fracture It is a mistake, for example, to treat a fracture of the shaft of the femur without initial manipulation but simply by the suspension of a weight from a skeletal traction pin in the bone with the object of correcting displacement gradually over a period of days or weeks Two or three weeks may elapse before it is recognised that reduction is imperfect; the weights may then be increased and there is danger of distraction; repeated adjustments are made and there is constant interruption, after long delay it may be found that operative reduction is necessary. Such a routine is harmful to the healing fracture because it disturbs the growth of granulation tissue and callus exactly at the time that freedom from disturbance is most important Frequent interruption, alteration of position, distraction of fragments, and late operative intervention, are all factors that cause delayed union[1] The purpose of treatment of any fracture should be to complete the final reduction of displacement within two or three days, and then to maintain the reduced position continuously and without interruption In the case of a fractured femur, displacement should be reduced by manipulation, and if necessary by heavy traction under anæsthesia, the limb then being immobilised in splints with just sufficient continuous traction to prevent redisplacement. Heavy continuous traction should be avoided, and under no circumstances must the fragments be distracted Similarly, fractures of the tibia must not be over-pulled If a fracture is unstable, and continuous traction is needed,

[1] Watson-Jones, R, and Coltart, W D "Slow Union of Fractures" *Brit J Surg*, 1943, 30, 260

not more than about 10 lb. should be used. Fractures that would otherwise unite in eight or ten weeks unite after distraction only in eight or ten months. Such delay is entirely unjustifiable; it would be much better to control instability and prevent redisplacement by internal fixation.

MECHANICAL REDUCTION AND SKELETAL TRANSFIXION

A technique of skeletal transfixion of bones, first developed many years ago by Lambotte,[1] has been reintroduced in the United States of America by Roger Anderson, Haynes, Stader and others.[2-5] Two pairs of obliquely placed pins are introduced, one pair into the proximal fragment and one into the distal fragment (Fig. 314). The pins are incorporated in a machine which reduces the fracture. Traction, angulation and rotation are controlled fluoroscopically. The pins are incorporated in plaster or joined by a steel bar—an "extra-skeletal bone" which is said to permit immediate mobilisation of joints and prompt weight-bearing.

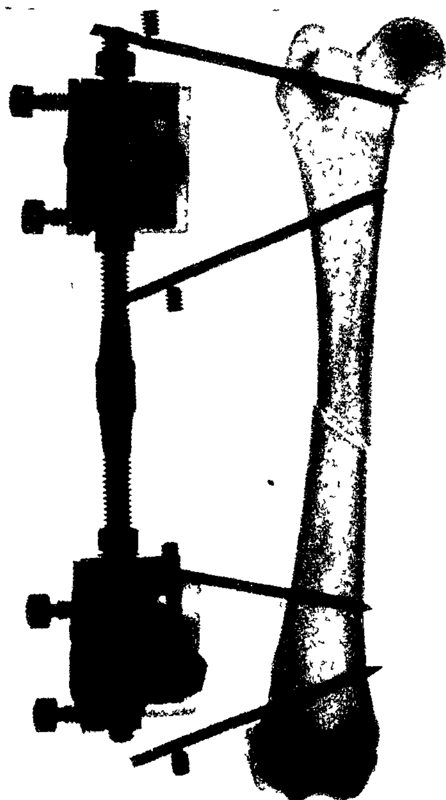


FIG. 314

Demonstrating the principle of skeletal transfixion by two pairs of obliquely placed pins incorporated in a steel bar that serves as an 'extra-skeletal bone." This particular splint is the type originally devised by Stader, a veterinary surgeon, for use in animals.

This is indeed an age of mechanisation and we should beware lest surgeons forget to use their hands, but still more should we beware lest surgeons forget to use their comprehension. It need only be asked how the knee joint can possibly be mobilised when the quadriceps is transfixed to bone by not less than four pins. That there may be a limited range of early movement is not doubted, but that this confers any ultimate benefit is not yet proved. Moreover, we know the dangers of low-grade infection round one pin track, and the dangers of low-grade infection round four pin tracks is four times as great.

[1] Lambotte, Albin. "Chirurgie Opératoire des Fractures." Paris: Masson et Cie, 1913.
[2] Anderson, R. *J. Bone Joint Surg.*, 1934, 16, 379.
[3] Haynes, H. H. *South. med. J.*, 1939, 32, 790.
[4] Shaar, C. M., and Kreuz, F. P. "Manual of Fractures—Treatment by External Skeletal Fixation." Philadelphia and London: W. B. Saunders & Co., 1943.
[5] Lewis, K., Breidenbach, L., and Stader, O. "Stader Reduction of Splint in treating Fractures of the Shafts of the Long Bones." *Ann. Surg.*, 1942, 116.

CHAPTER X

OPERATIVE REDUCTION OF FRACTURES

"The most unfortunate innovation in the treatment of recent fractures is routine exposure and reduction by open operation: thousands of lives have been sacrificed and many more have been crippled"—L. BOHLER[1]

"The contentions of Lane are sound and have proved beyond question that when indicated and well done, the operative treatment of fractures has given the best results"—O'NEILL SHERMAN[2]

Where is the truth? The truth is in the words "when indicated and well done" The surgeon who operates before he learns the principles of fracture treatment is responsible for crippling, the surgeon who has not even learnt to operate may be responsible for loss of lives Experience of the non-operative methods by which perfect results can be secured in the great majority of fractures is essential With this knowledge, and with an aseptic technique that can be relied upon, the operative reduction of certain fractures is justified On the other hand, the surgeon who plates a fractured femur by an indifferent technique and then puts the patient to bed with the limb supported on a pillow is not capable of treating such a fracture by any method, much less by operative methods.

Disadvantages of operative reduction—To infect a simple fracture by a careless operation is a major tragedy The most scrupulous aseptic technique is necessary The standards of asepsis that may be safe for abdominal surgery are unsafe for operations on bones and joints Forty-eight-hour skin preparation, ten-minute scrubbing up, complete screening of skin by stockinet or towels, no-touch technique, and absolute integrity of the whole theatre staff are the elements of this technique It is said that the "safest period" is the tenth to the fourteenth day, but there can be no question of relative safety or of degrees of risk With perfect precautions there should be absolute safety and no risk. Any day should be a safe day

Apart from the possibility of infection there are other disadvantages. The stripping of muscles and periosteum which interferes with the blood supply of the bones, and the dissemination and dilution of the fracture hæmatoma, may delay repair Furthermore, exposure of the bone may promote intermuscular adhesion formation and joint stiffness

Optimal time for operative reduction—The decision that a fracture requires treatment by a manipulation and plaster, b manipulation and continuous traction, or c operative reduction and internal fixation, should be made within a few days of injury It is not justifiable to try one method, then some days later another, after several weeks a third, and finally to adopt operative reduction as the last resort The principle of "timing of fracture

[1] Bohler, Lorenz "Treatment of Fractures" Bristol John Wright, 1935, 82
[2] Sherman, O'Neill. "Poor Results in the Treatment of Fractures" *Trans Amer med Ass. (Orthop Surg)*, 1922, 71

treatment" must be respected. There should be no interference with the healing process during the vital period from the second to the tenth weeks. As a rule the decision can be made when the fracture is first examined, and if an operation is required it should be performed at the ideal time, within a few days of injury Even if it is decided that one or more manipulations should be tried first, these manipulations should be completed, and the decision made, within three or four days



FIGS 315-316

The result of operative reduction of a fracture of the shaft of the femur by a surgeon who was obviously *untrained in the principles of fracture treatment* The operation was badly done, the plate was too short, no screw engaged the opposite cortex, the technique was deplorable, and the patient then lay in bed with the limb on a pillow! There is angulation, anchorage of the quadriceps, 90° drop foot and rigid clawing of the toes

INDICATIONS FOR OPERATIVE REDUCTION

There are four main indications for operative reduction and internal fixation 1) the probability that manipulative reduction will not succeed, or that it has been tried and failed, 2) the probability that manipulative reduction will not be maintained, or that redisplacement has actually occurred; 3) the probability that union will be slow; 4) the fact that non-union is already established

1 **Manipulation will not succeed**—The failure of manipulative reduction may arise from displacement of fragments by elastic retraction of muscles; displacement of fragments with interposition of soft tissues, displacement of fragments still unreduced many weeks or months after injury, or displacement of small and inaccessible intra-articular fragments

Elastic retraction of muscles—Fractures of the patella may be displaced by retraction of the quadriceps muscle, and fragments of the olecranon may be displaced by the triceps muscle These fractures are to be regarded as bone injuries incidental to the rupture of tendons which necessitate operative suture Other tendon ruptures or avulsions accompanied by fractures do not necessarily demand operative treatment Fractures of

the epicondyle of the humerus from avulsion of the flexor muscles of the forearm may often be treated successfully by simple flexion of the elbow Fractures of the anterior iliac spines by avulsion of the sartorius and rectus femoris call only for flexion of the hip Fractures of the tuberosity of the humerus with retraction of the supraspinatus may be treated by abduction of the shoulder Fractures of the base of the fifth metatarsal by the pull of the peroneus brevis require simple immobilisation in plaster

Interposition of soft tissues in fractures—Operative treatment is needed in fractures of the medial malleolus of the tibia with interposition of a flap of periosteum, fractures of the shaft of the radius with interposition of the pronator teres, and fractures of the shaft of the femur with interposition of muscle In fractures of the tibial malleolus the complication is shown by radiographic evidence of a persistent gap between the fragments In fractures of the shaft of the femur it is usually impossible to judge by radiographs alone that soft tissues are interposed; the important test is not only that manipulative reduction has failed but that it has proved impossible, even with strong lateral pressure, to bring the fragments into apposition and that throughout the manipulation no crepitus was elicited

Interposition of soft tissues in dislocations—Joint dislocations may be irreducible because soft tissue flaps are interposed In dislocations of the elbow joint the tendinous origin of the flexor muscles may be drawn into the inner side of the joint with a fragment of the medial epicondyle of the humerus, manipulative reduction cannot be successful until the flap of tissue is hooked out Similarly, reduction of dislocations of the knee joint may be obstructed by a flap consisting of the medial collateral ligament, part of the quadriceps expansion and the capsule of the joint, torn from the medial side of the femur and interposed between the articular surfaces. Unlike the corresponding dislocation of the elbow joint there is no radiographic proof of the interposition, but clinical evidence is available in the impossibility of replacing the bones in normal apposition no matter what strength is used, and in the dimpling of skin over the inner side of the joint when strong lateral pressure is applied from the pull of underlying tissues imprisoned in the joint Metacarpo-phalangeal dislocations may also be irreducible by manipulation because the torn capsule of the joint is buttonholed round the metacarpal head

Late unreduced fractures—When many weeks or months have elapsed since injury, manipulative reduction may be impossible because the fragments are joined in malposition by scar tissue and callus It is necessary to cut down on the fracture, freshen the bone ends, angulate the fragments into position or secure reduction by strong traction, and use internal fixation

Intra-articular fractures—Small fragments may have been chipped from the articular surface of a joint and necessitate excision—for example, marginal fractures of the capitellum, fractures of the head of the radius, and fractures of the femoral condyles Similarly, fractures of the tibial tuberosities may not always be amenable to manipulative replacement and it is sometimes necessary to open the joint and replace articular fragments

2. **Manipulative reduction will not be maintained**—Certain fractures are so unstable that redisplacement of the fragments is likely to occur as, for example, oblique and spiral fractures of the tibia even despite perfect initial



Fig. 317



Fig. 318

Established non-union of a fracture of the shaft of the humerus of twelve years' duration Ni operations had already been performed Union was secured by freshening the sclerosed bone end implanting cancellous bone chips cut from the ilium, and supplementing the external support of shoulder plaster spica with the internal fixation of an intramedullary nail.

reduction and careful immobilisation in plaster. The alternatives that are available are continuous traction and internal fixation. If the surgeon can be certain of the reliability of his aseptic technique, open reduction and internal fixation are fully justified and, indeed, are to be preferred, since methods of continuous traction often cause serious delay in the union of fractures of the tibia The same alternatives of continuous traction or internal fixation are available in fractures of the lower shaft of the radius with inferior radio-ulnar dislocation. In fractures of the upper shaft of the ulna with dislocation of the head of the radius (Monteggia fracture-dislocation), the instability is best controlled by operative fixation of the fracture of the ulna. In fractures of the shafts of the forearm bones it is usually wise to try conservative measures first, but if the fractures cannot be reduced perfectly, and there is proved instability, there should be no hesitation in using internal fixation in order to prevent late redisplacement.

3. **Union of the fracture will be slow**—The most striking example of a fracture that can be reduced perfectly, but in which slow union justifies internal fixation, is fracture of the neck of the femur Most of these fractures would no doubt unite in time if carefully moulded plaster spicas were reapplied whenever they became loose, and complete immobilisation was continued for the necessary period But union is often so slow that the necessary period might be as long as twelve months or two years, and it is unreasonable to handicap an elderly patient in a plaster spica for so long Internal fixation is obviously preferable In other fractures the likelihood of slow union is recognised only three or four months after injury Fractures of the lower shaft of the tibia and the humerus may show little union despite complete immobilisation for several months The fracture will certainly unite if immobilisation is continued long enough, but a bone-grafting operation may be performed as a time-saving measure It is to be emphasised, however, that the repair of a slowly uniting fracture will not be accelerated by simple open reduction and plating Plating of bones cannot be expected to play a part in the treatment of slow union, delayed union, or non-union ; bone transplantation is necessary

4 **Non-union is established**—When non-union is established operative reduction, bone grafting, and internal fixation are necessary Figure 317 is an example of a fracture that had already been treated by no less than nine operations ; they had failed, not because there was no apposition of the fragments, but because fixation had been imperfect After refreshing the fragments and inserting bone chips cut from the ilium, the external fixation of a shoulder plaster spica was supplemented by the internal fixation of an intramedullary nail (Fig 318)

ASEPTIC TECHNIQUE

The general standard of aseptic technique in operating theatres is still too low It is a grave reproach that a post-operative infection rate of as much as 2 per cent has sometimes been accepted with complacency. Infection of one clean case in a thousand is a disaster of the first magnitude. Even slight delay in healing, redness of skin, or any other sign of wound reaction, is evidence of failure. The ritual of asepsis can be learned only by

training and experience It must be acquired as a habit—subconscious, reflex, automatic—firmly ingrained in the minds of surgeon, assistant, theatre-sister and nurse. All must share the responsibility and all must know they share it[1] No single case of post-operative infection must pass unnoticed If a disaster has occurred an inquiry should be held every detail of technique should be reviewed , a skilled watcher may be appointed for a period to draw attention to faults on the part of the surgeon himself or of any member of his team Only then is the weight of responsibility understood fully

Theatre—One theatre should be reserved for aseptic cases Floors, walls, furniture and equipment should be phenolised daily. Windows must be heavily screened with gauze to prevent the entry of flies and dust Doors must be kept shut No one should enter in outdoor clothes or footwear, either on operating days or on visiting days , dust is the carrier of infection and every source of dust must be excluded In ideal circumstances the operating theatre should be air-conditioned, all entering air being filtered and purified , the temperature should be controlled in order to prevent the surgeon or his assistants from sweating. Ultra-violet light may be used to sterilise the air[2] It has been recommended not only that the air but also that the theatre walls, ceilings, and all equipment, should be sterilised by liberating formaldehyde in a hermetically sealed theatre before putting the air-conditioning plant in operation[3] These ideal conditions may not always be attainable, but much more can be done to control dust-borne infection than is now customary. We can stop all and sundry from walking in , we can stop the theatre from becoming the house surgeons' meeting place or the nurses' recreation room , we can bar entry to all who are not suitably clad

Staff and visitors—Every individual who enters the theatre must be clothed in sterile gown, cap, mask and cloth boots, this applying at all times and not only when operations are in progress Masks must cover the nose as well as the mouth, and they must include cellophane sheets interposed between the layers of gauze Many individuals who appear to be healthy are carriers of streptococci in the upper respiratory tract, and the use of impervious masks is of the greatest importance Talking in the theatre should be minimised , coughing and sneezing should be controlled The cap should be deep enough to cover the eyebrows, which often contain staphylococci The junior theatre nurse must not enhance her charm by allowing stray curls to appear The patient must not lie uncapped and unmasked, as so often is the case, and his coughing must be carefully guarded *Gowns must* be closely tied at the back , no unsterile clothing should be visible Nurses and visitors should stand back , they must never come in contact with instrument tables, theatre-sister, or surgeon

Patient—The site of operation, and a generous area above and below, should be prepared repeatedly for not less than twenty-four hours, and in the case of foot operations for forty-eight hours This long preparation is needed because two types of organisms exist in the skin—" transients " and " residents " Transient organisms are disposed of by a single washing

[1] Prevention of " Hospital Infection of Wounds " Medical Research Council War Memorandum, No 6 1941. H M Stationery Office
[2] Hart, D *Surgery* 1937, 1, 770
[3] Gudin, M *Ann Surg* , 1942, 115, 452.

with soap and water, resident organisms are more securely established and their eradication calls for repeated washing, cleaning with ether, and painting with antiseptics. The prepared area should be covered with at least two layers of sterile towel The outer layers are removed in the anteroom, and the inner layer only when the anæsthetised patient is in position on the operating table. It is often unrecognised, although it should be obvious, that blankets and pillows carried from the ward, teeming with bacteria, must in no circumstances go into the theatre with the patient. The patient should be transferred in the anteroom from a ward trolley to a theatre trolley with specially sterilised sheets, blankets and pillows which are the equipment of the theatre and never leave the theatre

Linen—Towels, side-sheets, stockinet, gowns, caps and masks must be sterilised by steam under pressure [1] Sterile linen should be taken from the tins by long-handled sterile "lifters," care being taken to avoid contact of the lifters, or of the nurse's sleeve, with the unsterile edge of the tin or its lid

Operating sisters, assistants and surgeons—Surgeons and nurses must at all times avoid contact of their hands with infected matter [2] Even before approaching the theatre their hands should be clean, their teeth clean, and their hair clean. They should change into sterile clothes, caps, masks and cloth overboots. For ten minutes by the clock they must wash fingers, hands and forearms, paying special attention to the sides of the nails, webs of the fingers and skin creases of the wrist The hands and forearms should be rinsed in antiseptic and dried on a sterile towel A sterile gown should then be put on, with care not to touch the outer surface of the gown or sleeves with the hands After the tapes have been tied by a nurse, a sterile back panel should be fixed in position by the surgeon in such a way as to cover the tapes and the gap between the margins of the gown which have been contaminated by tying This is important in lengthy bone-grafting operations, where in moving from one position to another the surgeon or sister may brush past an instrument tray When fingers and hands have been washed for ten minutes, and antiseptics have been applied, the skin is sterile, but it does not remain sterile Within half an hour resident bacteria, previously in the pores, are on the surface of the skin Sterile gloves must therefore be worn Even gloves do not afford full protection, because about 20 per cent are punctured at the end of an operation. Allowing for the hands of surgeon, assistants and nurses, there is at least one punctured glove in 75 per cent of operations Weed and Groves [3] recently found that one or more gloves had been perforated in 3,409 out of 4,549 consecutive operations Infected sweat inside a rubber glove is under pressure and it may actually be sprayed through a puncture hole One theatre epidemic was traced to a surgeon's hands carrying staphylococcus aureus [4] For this reason, even when sterile rubber gloves are worn, special precautions should be taken

[1] Cutler, E C, and others "Sterilisation and Aseptic Technique" *Inst Abstr Surg*, 1940, 71, 414

[2] **W Thelwall Thomas** of Liverpool always carried a pair of rubber gloves in his hip pocket and wore them when examining an ulcer, a sinus or an infected wound, the gloves were then re-sterilised and returned to his hip pocket ready for the next occasion He claimed that his hands were always sterile Thomas completed his medical training in Glasgow where he was influenced by Lister's teaching He was largely responsible for introducing aseptic technique to Liverpool hospitals and, with F T Paul of Paul's tube fame, was a pioneer of abdominal surgery He was the most dexterous surgeon I have known Nearly every operation was completed without a single blood stain on the side towels This was his pride, and no house surgeon who offended by allowing contact of a blood-soaked dab with the towels ever dared to offend again One of the greatest privileges of my life was to be Thelwall Thomas' house surgeon—the last before his retirement R W -J

[3] Weed, L A, and Groves, J L "Surgical Gloves and Wound Infection" *Surg Gynec Obstet*, 1942, 75, 661

[4] Devenish, E A, and Miles, A A *Lancet*, 1939, 1, 1088

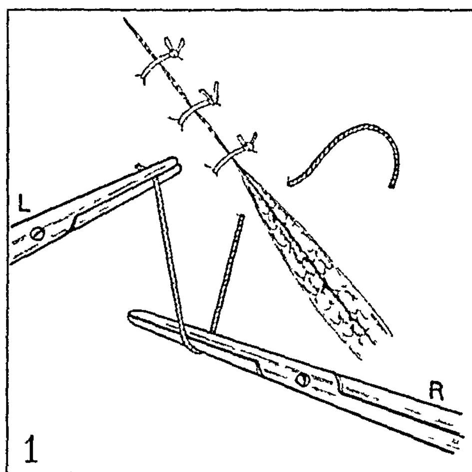


Fig 319

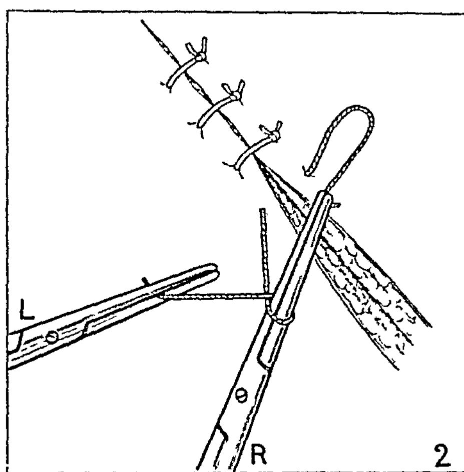


Fig 320

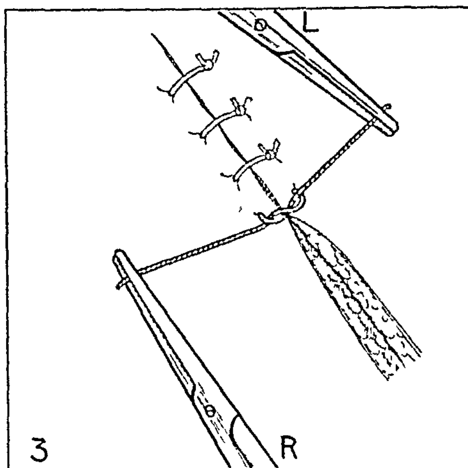


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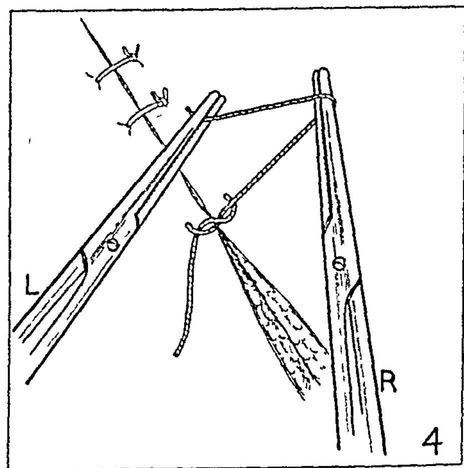


Fig. 322

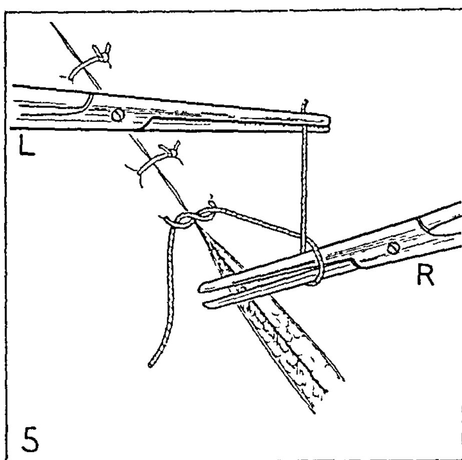


Fig 323

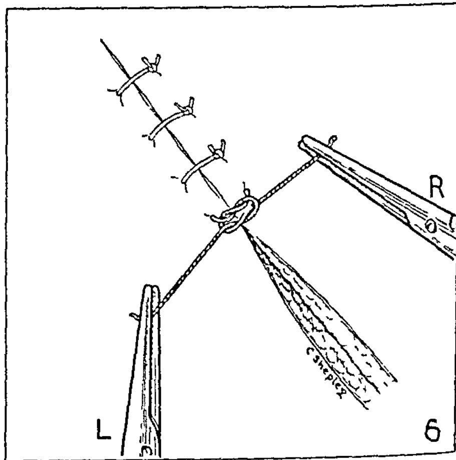


Fig 324

No-touch technique—tying sutures and ligatures with forceps

A surgeon should not operate on fractures unless he is a master of no-touch technique, including the tying of ligatures and sutures by two pairs of forceps After sterilisation, the thread or gut is never touched by hand With very little practice it is no less easy, and indeed I think more easy, to tie ligatures and sutures with instruments than by hand

in the handling of instruments, the arrangement of the surgeon's instrument tray and the adoption of no-touch technique.

Instruments—All instruments must be boiled for not less than ten minutes and reboiled between every operation The theatre-sister should not touch an instrument by hand but only with sterile forceps or lifters, needles should be threaded with forceps and passed to the surgeon in a needle-holder, swabs should be handed out with forceps. instruments should be sorted with forceps No one in the theatre—nurse, sister, assistant or surgeon—must ever touch the "business end" of an instrument such as the blade of a retractor If this should happen inadvertently, or if any instrument touches the patient's skin, it should be reboiled before use

Instrument tray—If instruments are left lying about, or are placed haphazard on the surgeon s tray, the handle end of one instrument is likely

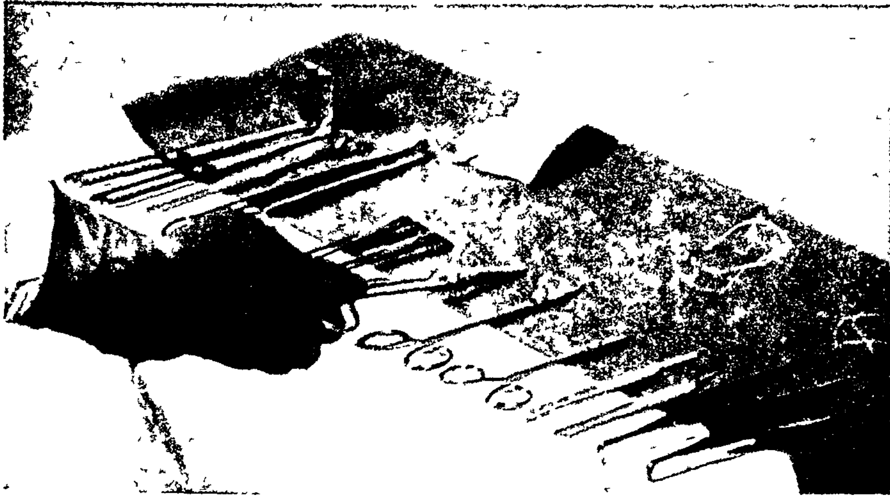


Fig. 325

No-touch technique—surgeon's instrument tray

Gloves are often punctured during operations The handles of instruments may therefore be contaminated, they must never come in contact with the "business ends" of other instruments The surgeon's tray is divided into two parts The business ends of instruments are always replaced on the coloured cloth which is never touched by hand, directly or indirectly

to contaminate the business end of another The surgeon's instrument tray should therefore be divided into two parts by means of a coloured cloth which is never touched by hand and never contaminated by contact with the handle end of an instrument Immediately after use, every instrument is replaced on the tray with its business end on the coloured cloth (Fig 325).

√**No-touch technique**—Before completing the arrangement of sterile towels by which to screen all surrounding areas, the site of operation itself should be covered with a layer of sterile stockinet fixed to the skin by sterile mastisol. It is then possible to palpate the region without danger of contamination of the surgeon's gloves, and to make an incision through the layer of stockinet without danger of contamination of tissues from the skin. Bleeding points should be secured with Spencer Wells artery forceps, and if ligatures are necessary they should be tied with two pairs of forceps, neither surgeon nor assistant ever touching the catgut by hand Swabs of dry gauze used for mopping the wound are not touched by hand but only

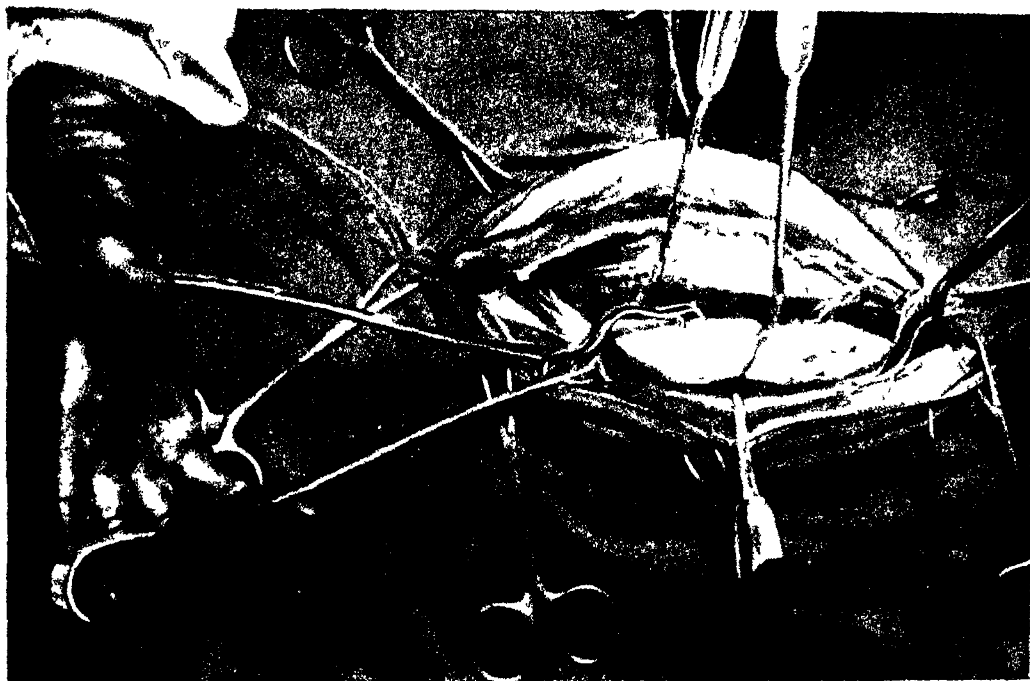


Fig 326

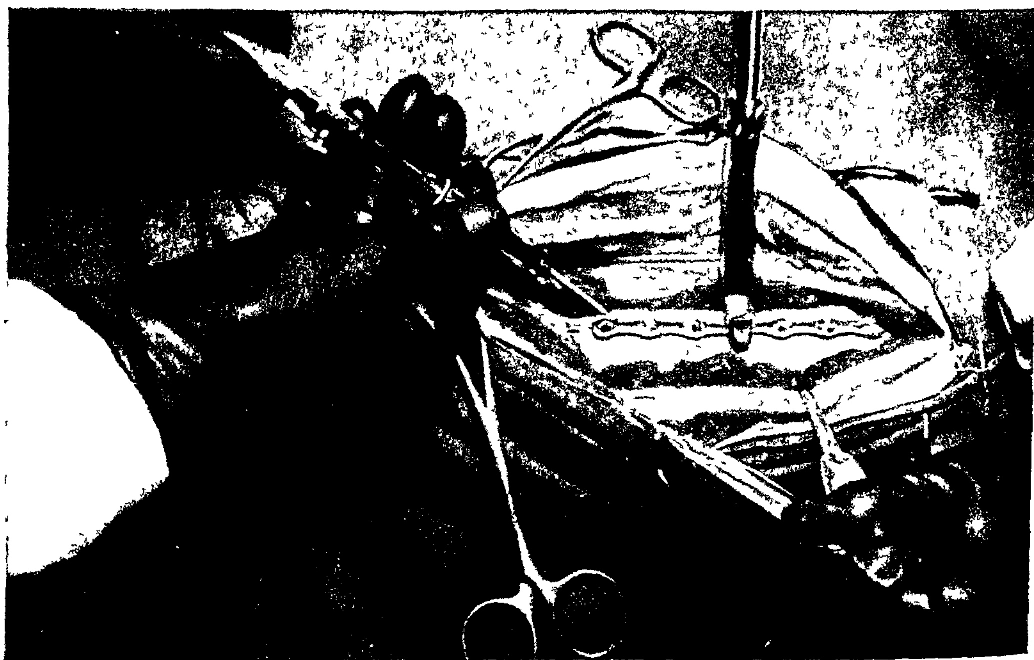


Fig. 327

No-touch technique—field of operation

Open reduction and internal fixation with plate and screws. Note that the patient's skin is completely screened with side towels, the surgeon's and assistants' hands are kept well away from the wound by long-handled forceps and retractors (Fig 326), the plate is held with special forceps and the screws with special screw-drivers so that neither are touched by hand (Fig 327).

with forceps; the assistant is never without a pair of forceps in one hand for this purpose Long retractors are used so that hands are well away from the wound. Needles threaded with forceps are held in needle-holders and sutures are tied with forceps Very occasionally it is advisable that the surgeon should palpate the tissues—for example, to examine the density of a nerve bulb, to determine the nature of a tumour, to explore the walls of an abscess cavity and be sure that every track is laid open, and in abdominal and thoracic surgery to examine the viscera and retract the tissues more gently than can be done with an instrument The orthopædic surgeon who is performing ten or twenty operations a week may find it necessary to put his hands into the wound every second or third month, the visceral surgeon may need the direct touch of his gloved finger more often; but for each of them this should be an event, and for each of them it is salutary first to rinse their gloves in antiseptic and saline. At least this will stop them, when they want to rest their weary limbs, from resting their hands in the wound.

Internal fixation—If a fracture has been reduced and it is proposed to use internal fixation with screws or plates, special precautions of asepsis should be observed Arbuthnot Lane[1] kept a small steriliser on an instrument table, with water on the boil throughout the operation. All drills, screws, plates or nails were left in this until required Screw-holders, plate-holders and screw-drivers were put in the steriliser with their handles projecting over the edge before they were actually used in the wound After use, these instruments were returned to the steriliser until required again The end of the screw-driver was plunged in the boiling water after the insertion of each screw If all non-absorbable foreign bodies, and all instruments which come in contact with them, are reboiled in this way immediately before use, the dangers of air-borne infection are minimised

Blood-borne infection—The aseptic technique that has been outlined aims at excluding infection from dust and air, instruments and towels, surgeon's and assistants' skin, and patient's skin It is also possible for a clean wound to be infected by organisms carried through the blood from a distant focus of infection Although this is possible, it is very rare indeed The incidence must be the same as the incidence of spontaneous infection of closed fractures, and how many surgeons have seen more than two or three such cases in a lifetime? It is, of course, wise to defer an elective operation if the patient is suffering from boils, tonsillitis or similar infections But the possibility of blood-borne infection can seldom be claimed as the explanation of post-operative infection If there is an obvious distant source of infection an elective operation should not have been performed, and if there is no obvious distant focus the possibility is very remote indeed

Lymph-borne infection—Although blood-borne infection is rare, lymphatic infection is common if operations are performed in the region of pressure sores or unhealed wounds It is not enough that the pressure sore is healed; bacteria remain in the lymphatics long after the skin is repaired It is not enough that the operation is performed at a distance from the recently healed pressure sore, the risk is grave if there is any possibility of lymphatic communication The operation should be deferred as long as possible, and certainly for not less than six or eight weeks

[1] Fairbank, H A T "The Non-touch Technique" *Brit med J*, 1942, 2, 388

METHODS OF INTERNAL FIXATION

The fracture is exposed, callus is cleared from the fractured surfaces to restore the original serrations, and the fragments are locked by angulating them into position Occasionally this is all that is necessary If there is firm natural stability of the fragments it may be possible to avoid the introduction





FIG. 328 FIG 329

Fixation by onlay bone graft

Recent fracture of the shafts of both forearm bones Manipulative *reduction is unsatisfactory (Fig 328). Remanipulation might improve* the position, but in view of the unstable type of fracture and the *likelihood of redisplacement, particularly of the ulna, internal fixation* is better This has been secured by onlay bone grafts with vitallium screws (Fig 329)

of foreign bodies Nothing is more unfortunate, however, than to perform an open reduction, decline the opportunity of ensuring perfect position by internal fixation and, three or four weeks later, find that redisplacement has occurred If there is doubt as to the stability of the fragments, it is better to use internal fixation The methods that are available include an autogenous onlay or inlay bone graft, a single obliquely placed screw, a plate and screws, or an intramedullary nail Other methods which are not recommended are circumferential catgut, wire or metal bands, and

intramedullary beef-bone or ivory pegs Whichever device of internal fixation is used, it is to be regarded as a suture of bone and not as a splint for the bone ; it supplements but does not take the place of external splints and plaster The factors that determine resorption of bone in the region of metal, and in particular the principles of bone resorption in response to compression when devices for internal fixation are not protected by external support, are considered in the next chapter



Fig 330



Fig 331

Fixation by inlay bone graft

Mal-united fracture shaft of radius (Fig 330) There is 90° of rotation of the fragments, the antero-posterior plane of the wrist coincides with the lateral plane of the elbow After operative reduction an inlay bone graft was used for internal fixation (Fig 331) Correction of angulation and rotational displacement restored radio-ulnar movement

Autogenous bone graft—A skilfully performed bone-grafting operation often provides sufficient internal support Although the fracture is relatively recent and may be in no danger of failing to unite, a bone graft has the great advantage of facilitating instead of hindering repair, and of being absorbed in the process For small bones, like the radius and ulna, an onlay bone graft should be used with vitallium screws (Figs 328-329) In the case of larger bones the surgeon may choose between onlay and inlay grafts, vitallium screws sometimes being needed even with inlays.

Obliquely placed screw—The less foreign material introduced in the region of the fracture the better Since the internal fixation is no more than a bone suture, which supplements external fixation by preventing shearing and rotation movements, oblique and spiral fractures can be dealt with satisfactorily by means of a single screw transfixing the fragments (Figs. 332-333) In the case of very long oblique fractures it is sometimes advisable to use more than one screw. The fracture is reduced and the fragments are held with a bone clamp The correct axis of the screw must be chosen carefully, as nearly as possible at right angles to the plane of fracture and through an equal thickness of bone on each side The track



Fig 332



Fig 333

Fixation by obliquely placed screw

Five weeks' old fracture of the shaft of the radius with inferior radio-ulnar dislocation This injury is very unstable Redisplacement within the plaster has been prevented by one vitallium transfixion screw

is drilled It is important that the drill should be exactly equal to the root diameter of the screw, if it is wider than this, the screw threads will not engage fully in the bone, for the $\frac{9}{64}$-inch Sherman screws that are usually used a $\frac{7}{64}$-inch drill is needed A screw of the correct length is selected after measurement of the length of the drill hole, and it is driven home snugly but not tightly To prevent subsequent loosening, the screw should be of non-corrosive non-electrolytic metal, such as vitallium It should be a self-tapping machine type of screw, not a pointed conical wood screw which engages less tightly at the point than at the head

Plate and screws—When the fracture line is horizontal one screw cannot transfix the fragments and, if for any reason bone grafting is not employed, a plate and not less than four screws should be used (Figs 334-335) The points to be observed in the operation are absolute asepsis and no-touch technique, accurate reduction of the fracture and the use of a good bone

clamp to grip the fragments, a strong plate of non-corroding metal; self-tapping screws of the same metal as the plate to prevent electrolysis; drill of the same dimension as the root diameter of the screws, drill holes perfectly centred in the holes of the plate, at least one screw on each side of the fracture long enough to engage the opposite cortex, screws firmly but not tightly driven home, suture of periosteum but not of muscles; complete external fixation by plaster casts

Encircling suture with wire or band—It is tempting to immobilise a spiral or long oblique fracture with encircling wire or a Parham's band. Perfect fixation can be secured but the technique is unsafe. The pressure



Fig 334



Fig. 335

Fixation by plate and screws

Mal-united fractures of forearm bones with overriding of radius and inferior radio-ulnar dislocation The radius required open reduction, and in view of the instability a stainless steel plate and screws were employed This is less satisfactory than accurate bone grafting but more effective than an imperfect graft

of the metal causes absorption of the underlying bone and refracture at this level has been reported many times Even a strong catgut suture tied tightly round a bone can cause sufficient bone absorption to produce an almost spontaneous fracture

Intramedullary ivory peg—The insertion of an ivory peg into the medulla of the fragments secures accurate apposition and guards to some degree against angulation, but the solid peg blocks an important source of granulation tissue growth and delays repair Moreover, it is an inadequate "bone suture" for it does not prevent rotatory movement of the fragments Small pegs are absorbed and disappear but large masses of ivory are incompletely absorbed and remain as an inert mass in the bone (Figs. 336-337)

Intramedullary nail—Different types of intramedullary nail have been employed by surgeons throughout the last fifty years Fractures of the neck of the femur were nailed in 1897 by Nicolaysen[1] and in 1916 by

[1] Nicolaysen, J *Nord med Ark*, 1897, 8, 1

Hey Groves[1] who also developed the technique of intramedullary nailing for fractures of the shafts of long bones He introduced nails through the greater trochanter for fractures of the shaft of the femur, and through the great tuberosity for fractures of the shaft of the humerus Success was limited Solid nails that plugged the medulla were no more successful than pegs of beef-bone or ivory Even the flanged nails that were devised by Hey Groves seldom succeeded because there was electrolytic corrosion of the metal, and the nails became loose The discovery of stainless steel and other non-electrolytic alloys had to be awaited, and it was not until the method was reintroduced by Smith-Petersen of Boston in 1931, after the development of stainless steel and vitallium, that intramedullary fixation became routine practice in the treatment of fractures of the femoral neck[2]





FIG. 336 FIG 337

Fixation by intramedullary peg

Internal fixation with massive beef-bone intramedullary peg Ten years later (Fig 337) the peg is still incompletely absorbed.

Intramedullary nailing of shaft fractures was reintroduced in 1939 by Kuntscher of Kiel[3] A long nail, V-shaped or trefoil in section, was introduced from the end of the bone across the fracture site without operative exposure of the fragments, usually with the aid of a preliminary guide wire and under fluoroscopic control For fractures of the femoral shaft the nail was introduced through the greater trochanter, for fractures of the humerus through the great tuberosity, for fractures of the ulna through the olecranon process, and for fractures of the radius and tibia through short incisions at the upper or lower ends of the bones In general, no external fixation was used and the nail alone was relied upon ; in lower limb fractures weight-bearing was permitted within two or three weeks[4] The technique was developed at the Finnish war-front in 1942 and thereafter adopted widely in Europe under the pressure of circumstances of war[4,8] A technique that permitted rapid discharge of patients from hospital was seized with enthusiasm at a time when there could be no certainty that long-stay hospital beds would be available, especially when the method involved no exposure of the fracture site and therefore reduced the dangers of bone infection which had been aggravated by lack of hospital facilities and good nursing Necessity, in this case, was the mother of so fertile an invention that sound judgment was threatened Kuntscher now believes that intramedullary nailing should be the standard treatment for every single shaft fracture of every long bone in every adult—high and low fractures

[1] Hey Groves, E W *Brit J Surg*, 1918, **6**, 224
[2] Smith-Petersen, M N *Arch Surg*, 1931, **23**, 715
[3] Kuntscher, G *Zbl Chir*, 1941, **68**, 857
[4] Rocher, C "L'enclouage medullaire des os longs" *Pr med*, 1945, **53**, 94
[5] Soeur, R "L'osteosynthèse au clou" Paris Masson et Cie, 1946
[6] Soeur, R *J Bone Joint Surg*, 1946, **28**, 309
[7] Soave, F *Arch ital Chir*, 1944, **66**, 271
[8] Vidal, F J *J Bone Joint Surg*, 1948, **30-B**, 735



Fig 338



Fig. 339

Un-united subtrochanteric fracture of the femur The patient had walked on it and worked for ten years, but with increasing pain and disability which ultimately made it impossible to go on working Radiographs in position of strain confirmed that there was only fibrous union; the appearance of bone union was from overlap of shadows There was marked bone porosis with "cyst" formation in the femoral neck



Fig 340

Fixation by intramedullary nail

The operation was most difficult but finally the fragments were replaced in apposition, the cysts were filled with bone chips from the ilium, and fixation by an intramedullary nail was used together with a plaster spica



Fig 341



Fig 342

Nine months' old fracture of the shaft of the femur, imperfectly reduced and unsoundly united, in which refracture occurred after a slight stumble (Fig 341) After open reduction the fragments were immobilised by means of an intramedullary nail together with a plaster spica (Fig 342) Sound union was gained in perfect position

of the shaft, transverse and oblique fractures, simple and comminuted fractures, fractures with delayed union, fractures with non-union, paralytic fractures, fractures from disease of bone, fractures from malignant tumours, and even compound fractures with infection, where the astonishing suggestion has been made that if the nail is left in place "it acts as a drain carrying pus to the surface"! Can we say more for the limitless enthusiasm of this pioneer than that he uses a nail thirty-six inches long, extending from the trochanter to the ankle joint, to immobilise an arthrodesed knee!

Intramedullary nailing has great value in the treatment of fractures of the upper shaft of the femur, Monteggia fracture-dislocations, and un-united fractures of the humerus (Figs 317-318) It may have other applications—but let us be sure that enthusiasm does not outweigh sound judgment.

CHAPTER XI

REACTIONS OF BONE TO METAL[1]

"The crying evil of our art is that much of our surgery is too mechanical There is a hankering to interfere which thwarts the inherent tendency to recover We want knowledge that will aid repair, not better mechanics."

These words, written sixty years ago by Hugh Owen Thomas, apply no less forcibly to-day when there is again a tendency to base the technique of bone surgery on mechanical considerations rather than on physiological principles Metal ligatures and pins have been used in surgery for more than three centuries but it is only in recent years, particularly since bone infection was partly controlled by the antiseptics of Lister and the antibiotics of Fleming, that plates, screws, pins and nails have been used so generally in the treatment of fractures It may be opportune, therefore, to review the reactions of bone to metal, and to consider them under four headings thermal, electrochemical, bacteriological and physical

THERMAL DESTRUCTION OF BONE

Developments in the technique of bone surgery have called for precision tools, motor-driven saws and high-speed drills Surgeons should, of course, be expert craftsmen and use efficient tools, but the material on which they work is not metal or wood—it is living bone, the cells of which are destroyed by heat For example, a graft was cut from the subcutaneous surface of the tibia with a high-speed motor saw and, three weeks later, cortical bone one centimetre from the site of removal of the graft was examined histologically (Fig. 343). It was dead—not because it had lost its blood supply but because it had been killed by the heat of a fast-revolving saw. If bone had been destroyed at such a distance there can be little doubt that the graft itself was also dead This does not mean that every graft must necessarily be cut with a chisel It will be



FIG. 343

Microsection of corticalis of the tibia cut, after three weeks, at some distance from the site of removal of a graft with a high-speed motor-saw (stained van Geisen, ×230) All spaces are empty, the bone has been burnt to death.

[1] Based on the Robert Jones Lecture delivered in the Royal College of Surgeons of England, December 1948, and the Hugh Owen Thomas Memorial Lecture given in Liverpool, November 1949

seen in another chapter that, with the exception of cells on the surface, all transplanted bone dies, and that cortical grafts, which are used primarily for internal fixation and the provision of scaffolding rather than for inherent vitality, may succeed even when surface cells die, when the graft is boiled, or when cadaveric bone is used The demands of accurate shape and fit may therefore justify the use of an electric saw even despite the effects of burning But when accurate carpentry is not essential, and the survival of endosteal and subperiosteal cells is more important, as, for example, when cancellous chip grafts are transplanted to fill a bone defect, a gouge or chisel should always be used in preference to a power-driven tool

The injury caused by a high-speed drill in preparing the track for a screw is not recognised sufficiently Such drilling involves not only the removal of a core of bone $\frac{1}{8}$ inch in diameter and its replacement by metal but also the destruction by heat of a surrounding area of bone which regenerates only after many months (Fig 344) Every hole so driven, and every screw introduced, is a source of weakness It is true that internal fixation often makes it difficult to avoid the use of screws In the treatment of congenital pseudarthrosis of the tibia, grafting operations usually failed in former years because reliance was placed on bits of catgut tied round the graft in a manner that would have been laughed to scorn by any carpenter, and it was only when double onlay grafts were secured with mechanical efficiency by four transfixion screws that the problem was solved, so that success is now achieved in nearly all cases instead of failure in 80 per cent Nevertheless, the occasional necessity for screws is an evil necessity In the case illustrated in Figures 346-349, sound union of a congenital pseudarthrosis of the tibia had been gained by double onlay grafting, but refracture occurred after several months through one of the screw tracks and recurrence of non-union was prevented only by prompt removal of the metal with the insertion of cancellous bone grafts and prolonged external fixation



FIG. 344

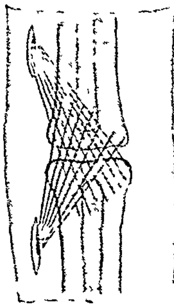


FIG 345

SIX months after removing screws from a grafted fracture of the radius (with excision of the lower end of the ulna), the drill holes are still obvious because surrounding bone has been burnt (Fig 344) In the treatment of non-union of fractures such drilling is obviously futile (Fig 345)

If one screw is needed, one alone should be used An onlay graft must usually be fixed with four screws, two in each fragment, but no more than four should be used, and quite often three or even two screws may suffice for an operation that is not an internal "fixation" but only an internal "suture" Every time that a surgeon prepares to drill a bone for the insertion of a screw he should pause to consider whether it is necessary



Fig 346



Fig. 347



Fig 348

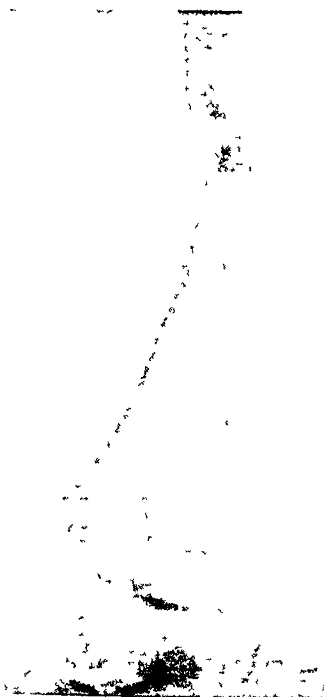


Fig. 349

Congenital pseudarthrosis of the tibia (Fig 346) treated by double maternal onlay grafting with four vitallium screws Several months after union, secondary fracture occurred through the site of one drill hole (Fig 347) The screws were removed and cancellous bone grafts inserted (Fig 348) The refracture then united and was soundly consolidated (Fig. 349)

Certainly we should be ashamed of the illustrations that blacken the pages of many journals with massive shadows of metal The use of any foreign body is an acknowledgment of our limitation Even difficult comminuted fractures can often be treated successfully with a single screw (Figs 350-351) It is true that such economy demands greater judgment and skill than the blind insertion of one screw after another, and, moreover, the satisfaction of operative craftsmanship must often be denied Consider the remarkable case shown in Figures 352-354, in which it was found possible to introduce eighteen screws and two plates into one simple fracture of the tibia More bone was destroyed by the operation than by the original injury, and the







FIG 350 FIG 351

If one screw will suffice, no more than one screw should be used. This is often possible even in difficult fractures as, for example, in the case treated by Mr Butler, of Montreal, while in the orthopædic service of the Royal Air Force during the recent war

victim sustained a series of spontaneous fractures, first at one drill-hole and then at the next only after three years could he step off a pavement with some assurance that his shin would not crack beneath him The purpose of this fantastic operation had been to make internal fixation supremely effective: but the technician—and that is a better name for him than surgeon—forgot that he was dealing with living bone

The destructive effect of high-speed drills must also be recalled in considering the operation of drilling sclerosed bone in the treatment of old un-united fractures (Fig 345). Sclerosis of fractured surfaces is a barrier to cellular activity and there is need to refreshen the bone, but it is obvious that cellular growth is not initiated when a high-speed drill is used The impotence of burnt bone is no better than the indolence of sclerosed bone

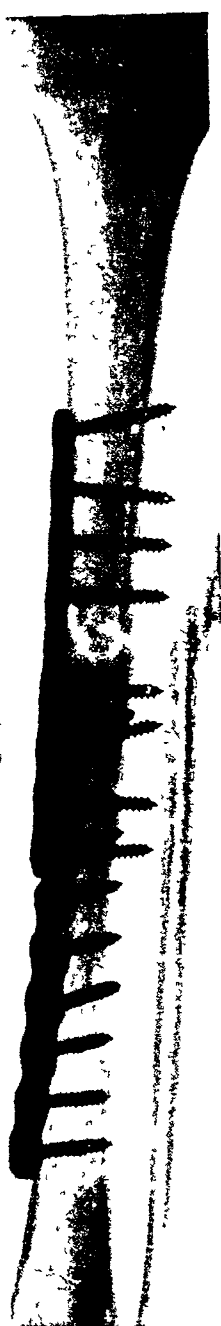


Fig 352

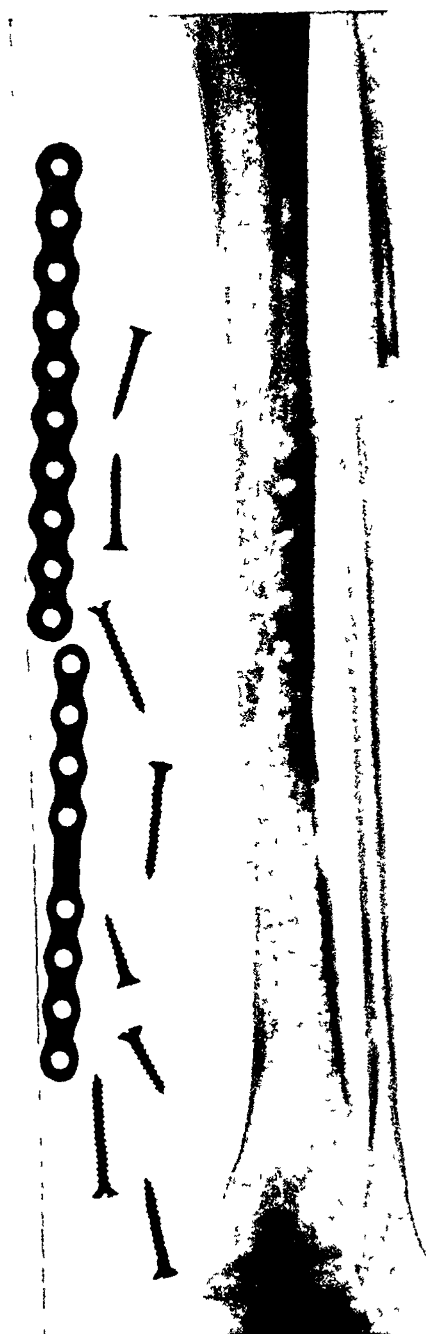


Fig 353

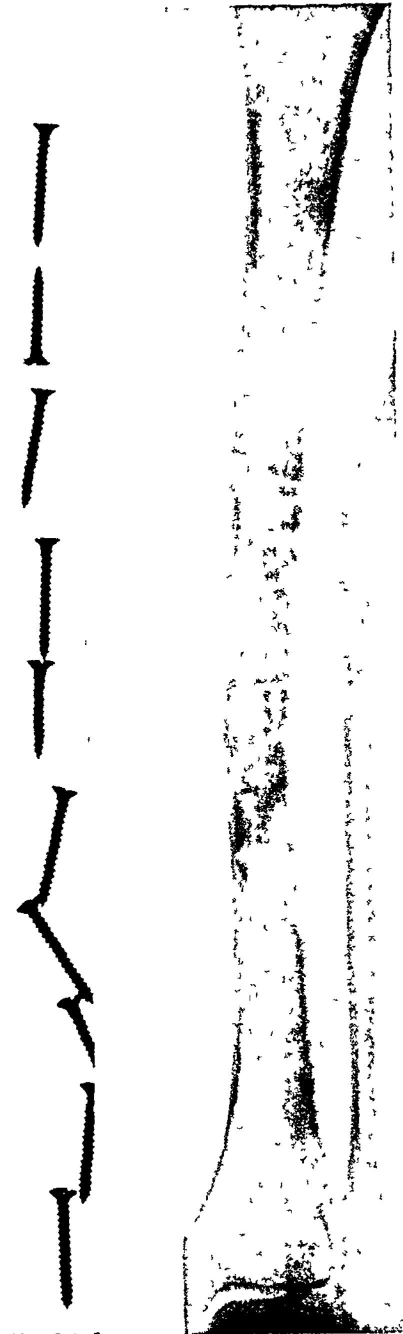


Fig 354

A technician, who called himself surgeon, found it possible to introduce eighteen screws and two plates into one simple fracture of the tibia (Fig 352) More bone was destroyed by the operation than by the original fracture Thereafter the unfortunate patient sustained five spontaneous fractures through the sites of drill holes (Fig 354) Only after three years could he step off a pavement with some assurance that his shin would not crack beneath him The joys of operative craftsmanship must not be indulged the driving of screws with power-driven tools is great fun, but it is a pleasure that must be denied

ELECTROLYTIC DESTRUCTION OF BONE

Fifty years ago, when the treatment of fractures by internal fixation was developed by Arbuthnot Lane, Lambotte and Hey Groves,[1] screws often became loose within a few months and there were many failures. Lane believed that bone resorption was always due to infection and declaring that "rarefying osteitis in plain English means dirty surgery" he developed a non-touch technique which gave him success far greater than that achieved by others Nevertheless, bone resorption still occurred and often continued for many years after union of the fractures (Figs 355-357). For a long time this was attributed to toxicity of the metals [2]

In 1934 Menegaux and Odiette [3] suggested that bone resorption round metallic foreign bodies might be the consequence of electrolysis rather than toxicity Orsos had already observed electrical currents when metals were implanted in bone In immobilising a fracture of the humerus with an aluminium plate and brass screws he had noted strong contraction of the extensor muscles of the forearm when the radial nerve came into contact with the metals More recently a valuable contribution has been made by Venable and Stuck [3] who have proved that electrolytic resorption occurs whenever dissimilar metals are used In earlier days, plates and screws were made of electroplated iron, brass, phosphorus-bronze, aluminium-bronze and copper-nickel alloys It was not realised that human saline fluids and organic acids serve as electrolytes, and that pairs of dissimilar metals set up batteries comparable to those used for lighting an electric bulb or ringing a front-door bell Ions were transferred from one pole to the other and, in the same way that there may be corrosion in the plates of any battery, there was corrosion of the bone screws and nails. Figure 358 shows a steel nail used to supplement open arthrodesis of the hip joint with a bronze cap placed over the head, in which a large part of the nail dissolved and disappeared

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Fig 355



Fig 356



Fig 357

Fracture of the radius plated in the 1914-18 war (Fig 355) Thirty years later, electrolytic resorption of bone almost resembled an osteoclastoma After removal of the plate and screws the bone regenerated (Fig 357) *(By courtesy of Mr L Gillies, Ministry of Pensions Hospital, London)*



Fig 358

A steel three-flanged nail used in arthrodesis of the hip joint was secured with a bronze cap over the head Ionisation between the metals caused solution of the nail

Non-electrolytic alloys—Electrolysis cannot occur when a single pure metal is used; but such elements as gold, silver, lead, tantalum and titanium have not the strength needed for the internal fixation of bones, and an alloy is essential. If the alloy consists of elements that are remote in the electrolytic series there is electrolysis between them; but if the elements lie so close that there is little potential difference the alloy is inert. This requirement is satisfied by vitallium—an alloy of cobalt 65 per cent, chromium 30 per cent, molybdenum 3 per cent., and manganese silicon carbon 2 per cent.—and also by 18/12 stainless steel in which there is 18 per cent chromium and

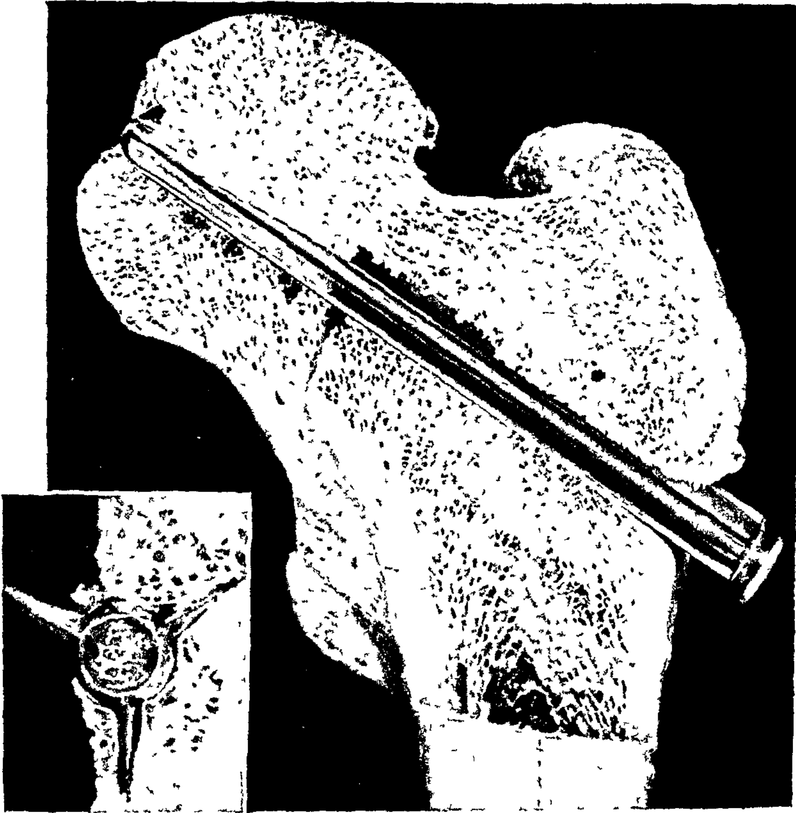


FIG 359

Non-toxic and non-electrolytic alloys do not cause resorption of bone. Fracture of the neck of the femur immobilised by means of a stainless steel nail. The point of the nail at the fovea, seen in the inset, shows that cancellous bone may grow even within the lumen of the nail.

12 per cent. nickel. There are, of course, fifty or more types of stainless steel and earlier varieties often corroded, but 18/12 S Mo steel is free from electrolytic reaction and is no less safe than vitallium[1] (Fig 359).

[1] To surgeons who spend their lives offering freely all they know the patents associated with these alloys have been a source of some annoyance. None has been the subject of a wider patent, and, at the same time, a greater publicity campaign, than vitallium. Many surgeons still feel unsafe unless they have screws and plates of this alloy despite the fact that it cannot be machined and must be cast, so that flaws of casting often cause fracture of the metal, but they may be reassured. Stainless steel, as now used by the best instrument makers, polished without iron filings by the process of "reverse-plating" which polishes the lumen as well as the surface of cannulated nails, is entirely free from electrolytic reaction and is safe in bone surgery. The surgeon should, however, confirm from his instrument maker that the steel he is using is in fact 18/8 or 18/12 S Mo. steel with a composition of

Chromium	17-20 per cent	Carbon	0·08 per cent max
Nickel	10-14 ,,	Silicon	·75 ,, ,,
Molybdenum	2-4 ,,	Phosphorus	0·03 ,, ,,
Manganese	2 per cent max	Sulphur	0·03 ,, ,,

Rockwell hardness, 30°-35° C.

Extrusion of metal by electrolysis—When three-flanged nails were first used in the treatment of fractures of the neck of the femur they were often extruded from the bone Some surgeons attributed this to impaction of (1) the fragments, and others to avascular necrosis of the femoral head, but (2) there can be little doubt that it was a positive expulsion by the pressure (3)





Fig 360 Fig 361

Fracture of the femoral shaft treated by intramedullary nailing with extrusion of the nail from pressure of corrosive products accumulating between nail and bone The nail was punched back and fixed with a cross screw

of corrosion-products of the metal accumulating between it and the bone. Similar expulsion is often seen when fractures of the shafts of long bones are treated by intramedullary nailing Figures 360-361 show extrusion of a medullary nail from the shaft of a femur When an incision was made in the trochanteric region to drive the nail back and fix it with a cross-screw, and the fibrous capsule around the nail was divided, there was a spurt of

fluid under pressure In one such case extrusion of a nail from the shaft of a femur was first recognised when the patient complained of pain in the chest, and it was found that the nail was in contact with the lower ribs! In another case a nail was extruded from a fracture of the shaft of a humerus and by impact against the acromion caused such pain on attempted abduction of the shoulder, even before the fracture was consolidated, that it was thought advisable to excise the acromion

BACTERIOLOGICAL DESTRUCTION OF BONE

Dirty surgery still goes on Closed fractures are still being infected at open operation—and there can be no greater disaster than that. It is more than fifty years since we were taught non-touch technique in the internal fixation of fractures, and it might be thought that progress would have been made in fifty years On the contrary, our surgery is no less dirty than it was Many surgeons claim that the technique is unnecessary, but if they were honest they would probably admit that they are too lazy to master it There is overwhelming evidence that infection of operation wounds arises from contamination by the hands of surgeons or their assistants, and that these infections cannot yet be prevented by antibiotics There is no difficulty whatever in achieving the internal fixation of fractures by non-touch technique, and no surgeon is at liberty to neglect it unless he can declare that neither he nor any member of his team has ever infected a closed fracture at operation If this standard was relied upon, non-touch technique would be almost universal

A metal plate, screw or nail is a sequestrum—A basic principle that has long been recognised is that infection of bone cannot heal until all sequestra have been resorbed, discharged or removed, and that union of a fracture is delayed so long as infection continues The first aim of treatment must be the healing of infection Only when infection is healed can bone be layed down, and only then can the fracture unite. It is a confusion of principles to say that plating is justified in the treatment of infected fractures by the need for immobilisation (Figs 362-363) It is true that healing of infection is aided by immobility, but it is not aided by the immobility that depends on buried metal which itself aggravates infection. A sequestrum may consist of a fragment of dead bone which must be removed—or it may consist of a foreign body such as a plate or screw which also must be removed, certainly it must not be introduced Thirty years ago metallic internal fixation of infected fractures was defended on the grounds that infection could be controlled by Carrel-Dakin antiseptic irrigation of open wounds in which the plates lay exposed More recently it was suggested that penicillin might control the infection It was then submitted that plating was justified if the metals were inert, though how any metal could be more "inert" than dead bone passes comprehension Finally it is now proposed that metal is safe if it is introduced at a distance and driven across the infected site by the technique of intramedullary nailing It has even been said that such a nail is useful "in carrying infection to the surface" What a travesty and distortion of surgical principles! In no circumstances should screws, plates or nails be used in the treatment of actively infected fractures; and even when infection is recently healed, but potentially





FIG 362 FIG 363

A patient sustained an open fracture of the shaft of the tibia The wound was excised by a surgeon who believed that the risk of infection was not very great and argued that, in any event, the best defence against infection was complete immobility of the tissues. He therefore plated the tibia and introduced six screws The wound broke down and infection persisted for months, with continued discharge from sinuses (Fig 362) The plate and screws were then removed together with small sequestra (Fig 363) Infection persisted until further sequestra separated, including the whole of the middle third of the tibia When, finally, the wound healed there was a four-inch gap of bone to be bridged, filled with dense avascular scar tissue which was still potentially infected

The reconstructive bone operation, in which cancellous bone chips alone could be used with no cortical bone and no metal, was difficult Yet if the original wound excision had not included the burying of foreign bodies which serve as sequestra the infection would have healed within two or three weeks and reconstructive surgery would have been easy There is no justification for metallic internal fixation of infected, or potentially infected, fractures No matter how " inert " a metal may be, it is a foreign body that must be removed Certainly it must not be introduced

active, internal fixation with metal should be considered only with great caution.

Recurrence of quiescent infection in response to metal—*Fractures with* recently healed infection are better treated conservatively than by operative internal fixation because the screws may cause recurrence of persistent grumbling infection which delays or even arrests union. Evidence of such

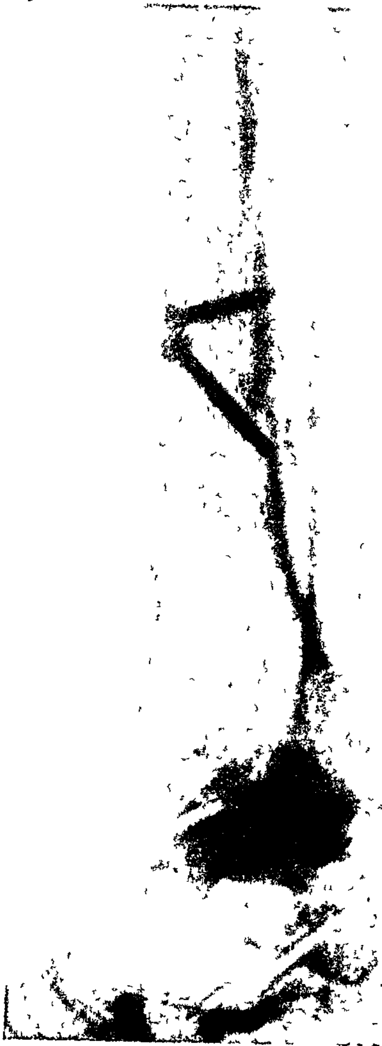


FIG. 364



FIG. 365

After the use of vitallium screws for the internal fixation of an open fracture of the tibia there was bone resorption round the screws without sinus or fever (Fig. 364). This in itself was evidence of low-grade infection Even five months after removal of the screws union was unsound (Fig. 365); it consolidated only after immobilisation of the limb for twelve months

low-grade infection is seen in the resorption of bone round one or more of the screws, and very often this occurs even when there is no clinical evidence of an abscess or sinus, or of acute febrile reaction *If the metal is non-electrolytic, and there is no compression of bone arising from the use of internal fixation without external support, radiographic evidence of bone resorption round a screw is itself evidence of infection* The foreign body must usually be removed before union of the fracture can be consolidated (Figs 364-365).

When a fracture with recently healed infection is un-united and there is an indication for bone grafting, it is much safer to use cancellous bone chips than whole-thickness cortical grafts Onlay grafts with screw fixation should certainly be avoided. Even without screw fixation the introduction of compact bone is unwise, and this is no less true of the technique recently proposed by Phemister in which the fracture is exposed from the side opposite to that of the sinuses. No matter which side of a bone is exposed, the field of lymphatic spread of infection is invaded and the danger is the same Neither metal screws nor compact bone should be used Reliance should be placed on cancellous chip grafts in which the bone trabeculæ are so thin that they are resorbed and regenerated without sequestration



Fig 366



Fig 367

"We have no internal fixation. We have only internal suture."

Figure 366 shows a subtrochanteric fracture of the femur treated by internal fixation without external support It is obvious that the internal fixation was hopelessly inadequate, the screws quickly loosened and the fracture failed to unite Figure 367 shows a fracture of the shaft of the tibia treated by internal fixation without external support The internal fixation was mechanically perfect, but the bone was again resorbed because it was tightly compressed, and this fracture also failed to unite (*Fig 367 is published by the courtesy of Dr Coonse, of Boston*)

PHYSICAL DESTRUCTION OF BONE

There can never be permanent union between metal and bone It is true that living bone can survive in close proximity to metal if there is no electrolysis or infection, but that is not to say that bone and metal can ever unite—which is what many surgeons have tried to achieve in the fixation of osteo-arthritic hip joints by nails, and the replacement of femoral heads by metallic prostheses (Fig 371) As soon as the junction between metal and bone is submitted to pressure, the bone resorbs and the metal loosens This

has long been known - Parham's bands, which tightly encircled spiral fractures of the shafts of long bones, achieved perfect fixation for the first few weeks, but late re-fracture at the site of compression occurred so often that the method was abandoned. Even sutures of kangaroo tendon tied tightly round the shaft of a bone may cause secondary fracture. Any attempt to rely solely on the metallic internal fixation of fractures must therefore be viewed with caution Figure 366 shows a subtrochanteric fracture of the femur treated by a plate and screws without the protection of splints or



FIG 368

Fracture of the shaft of the femur treated by intramedullary nailing with early weight-bearing and no external support Bone cannot withstand the pressure of metal, it is resorbed wherever pressure is applied This fracture failed to unite.

plaster, the metal soon loosened and the fracture failed to unite. It will be claimed that with fixation so inadequate loosening was to be expected; the screws did not even engage the opposite cortex Let us then consider an example of fixation which, from a mechanical point of view, was adequate. Figure 367 shows a fracture of the tibia secured with two plates, six bolts and twelve nuts The mechanics of the operation was perfect but, in so far as compression of bone was greater, the resorption was more rapid and the non-union no less certain, nearly all bone between the tightly bolted plates disappeared Even the wide surfaces of bone apposition afforded by an intramedullary nail cannot resist the resorption of physical compression. Figure 368 shows a nailed fracture of the femoral shaft in which the patient



FIG 369

Attempted fixation of the hip joint by nail alone It was bound to fail Bone resorption occurred both in the pelvis and the femoral neck wherever the stress of movement caused pressure of the nail on the bone



FIG 370



FIG 371

This intra-articular arthrodesis of the hip had been immobilised in plaster for three months but it was not enough Union was unsound and the nail could not protect it (*By courtesy of Mr Norman Roberts*) Bone reacts to the compression of metal by resorption Does this not mean that all attempts to replace the head and neck of the femur by a metal or plastic prosthesis must also fail because there can be no permanent union between bone and metal ? (Fig 371.) What will be the best treatment when the metal loosens in the bone ?

was allowed movement and weight-bearing within a week of operation The fracture failed to unite and there is clear evidence of resorption where the bone was compressed by the nail.

To my regret, I once advocated fixation of osteo-arthritic hip joints in elderly patients by the simple insertion of a three-flanged nail It was believed that these old people could not withstand the shock of an open arthrodesis, and it was hoped that the fixation of a nail alone might suffice (Fig. 369). It should have been obvious that there could never have been permanent success. Resorption of bone from compression, with loosening of the nail, was inevitable, and experience confirmed this so quickly that the method was soon abandoned But, despite repeated denials, the technique is still



Fig 372



Fig 373

Unsound union cannot be protected by metallic internal fixation Either the metal breaks or the bone breaks In this case the metal broke Despite intra-articular arthrodesis, insertion of cancellous bone chips, and protection in plaster for three months, union was unsound and the protection of a nail was insufficient for consolidation to be completed

being attributed to me, and even more massive nails and more efficient engineering devices with cross-pins and lag-screws are still being devised. How much more difficult it is to withdraw an observation than to make it! So imperfectly can mechanical internal fixation be relied upon that even after open arthrodesis of the hip joint, with all the precautions of removing articular cartilage, inserting cancellous bone grafts, and immobilising the joint in plaster for several months, a three-flanged nail is still incapable of protecting unsound union We have often been tempted to hope that the full protection of external fixation might be discarded after a limited period, and that early union would then progress to final consolidation under the safeguard of metallic internal fixation But this is not true Unless union is already sound, metal that is not strong enough breaks (Figs 372-373); and if it is strong enough the young bone breaks (Fig 370) We cannot

escape the conclusion that metallic internal fixation is no more than a supplement to the external protection of splints or plaster. In the words of Bishop Mumford of Indianapolis: "We have no internal fixation; we have only internal suture"

The same principle is demonstrated in two cases illustrated in Figures 374-378. In the first case, intra-articular arthrodesis was performed for a congenital dislocation of the hip joint. When fusion was found to be unsound an ischio-femoral fusion was attempted, but again, external support was discontinued too soon A solid blade plate was then used in an effort to provide internal fixation by which consolidation might be completed But "we have no internal fixation" no matter how massive the metal may be The blade plate broke (Fig 375) Exactly the same problem is illustrated in





Fig. 374 Fig 375

The use of a very solid blade plate, to complete the consolidation of an unsoundly united ischio-femoral arthrodesis, failed The plate broke "We have no internal fixation, we have only internal suture"

Figures 376-378 A patient with degenerative osteo-arthritis of the hip joint was treated by intra-articular arthrodesis with denuding of all articular cartilage and the internal fixation of a three-flanged nail supplementing the external support of a plaster spica Final consolidation failed because external support was discarded too soon The best treatment would have been the inlaying of a stout tibial graft between femur and ilium, packing the gaps with bone chips, and continuing the immobilisation in a plaster spica Instead, an ischio-femoral arthrodesis was attempted This too failed to consolidate quickly The surgeon was then faced with an unsoundly consolidated hip and an unsoundly consolidated ischio-femoral fusion (Fig 376) He thought that the protection of a blade plate and screws would be enough to complete the consolidation of both (Fig 377) But "we have no internal fixation; we have only internal suture" The screws broke as is shown in Figure 378



Fig 376



Fig 377



Fig. 378

Failed arthrodesis of the hip joint treated by ischio-femoral arthrodesis which also failed to consolidate quickly (Fig 376) It was thought that the protection of a blade plate and screws might suffice to complete the consolidation of both (Fig 377) But unsound union of bone cannot be protected by metal The screws broke (Fig 378).

FALLACY OF THE "COMPRESSION FACTOR" IN ACCELERATING THE UNION OF FRACTURES

Compression encourages union only in promoting immobilisation

The whole modern trend of fracture treatment is based upon the assumption that bone formation is encouraged by compression, and it is accepted almost universally that the union of lower-limb fractures is accelerated by weight-bearing compression. Many patients are forced to their feet at an early stage in the hope that osteogenesis will be stimulated thereby. Even more efficient methods of impacting and compressing the bones are now being developed. For the fractured neck of the femur we are urged to discard three-flanged nails in favour of lag screws which compress the fragments. For fractures of the shafts of long bones we are told to use slotted plates so that screws fixed in the bone can slide in the slots and allow continued impaction. Danis of Brussels has devised plates with special springs which push the bones against each other. Albert Key of St Louis wrote: "I started using positive pressure over twenty years ago. I put pins in, apply the turnbuckle and clamp the bones together." George Eggers of Texas claimed that the "contact compression factor" was fundamental in securing union and that with its aid fractures united in rats in seventeen days, whereas without it they did not unite at all.[1] Charnley of Manchester claimed even more when he said that by putting transfixion pins in each fragment, and clamping them together with a compression force of 80 lb. to the square inch, he could achieve "clinical union" as early as twelve to fourteen days.[2]

Disbelieving at once that any fractured human bone ever did or ever will unite in twelve days,* we must still consider the view, held so widely, that osteogenesis is stimulated and accelerated by forcible compression—a view that is surprising when we recognise the certainty with which the pressure of plates and screws causes bone resorption and not bone formation. It may be suggested that compression of bone by metallic foreign bodies is not comparable to the compression of "physiological forces within normal tolerance." Let us then consider physiological forces. When an aortic aneurism beats upon the spine it is surely a physiological force—it is no more than normal blood pressure, and yet invariably it causes resorption of bone. This is, of course, an intermittent pressure, though why an intermittent attack should be more dangerous than a continued attack is not clear. But consider the effects of continued pressure. A cyst of the meniscus which expands beneath the lateral ligament of the knee joint causes resorption of the tibial tuberosity by slow and steady pressure. A simple ganglion pressing on bone causes resorption.[3] Even if the spinal cord is pulled against the vertebræ by the contracting scar of an avulsed nerve root, the effect of pressure is felt more quickly by the bone than by the sensitive spinal cord, and the bone is resorbed.[4]

[1] Eggers, G. *J. Bone Joint Surg.*, 1949, **31-A**, 693.
[2] Charnley, J. *J. Bone Joint Surg.*, 1948, **30-B**, 478.
[3] Fisk, G. R. *J. Bone Joint Surg.*, 1949, **31-B**, 220.
[4] Penfield, W. *J. Bone Joint Surg.*, 1949, **31-B**, 40.

* These claims are misleading. When Charnley refers to "clinical union" he does not mean what most of us mean by this phrase—namely, that although union is not yet finally consolidated it is firm enough to submit safely to all ordinary stresses even without external protection or support. He only means that the fragments are "sticky"—and this is of course achieved in very many fractures whether compressed or not.

How small then is the physiological force of compression to which bone responds by *resorption*? Orthodontists can move a tooth lingually, labially or laterally; they can sink it into the alveolus or lengthen it out of the alveolus, they can move it in any direction by the simple application of elastic pressure. Within thirty-six hours of pressure being applied there is histological evidence of osteoclastic resorption in every area of compression. Is this the consequence of a force of 80 lb. to the square inch; or is it no more than the pressure of arterial blood? It is, in fact, a force so slight that no more than 20 mm. of mercury is enough—a pressure less than that of the capillary circulation. There can be no doubt that compression of bone is a destructive force.

Many surgeons have jumped to the conclusion that because distraction of bone fragments is known to delay the union of fractures, the opposite force must have the opposite effect. The fallacy is exposed by the studies of orthodontists. With a force of not more than about 20 mm. of mercury, bone is resorbed in areas of compression and laid down in areas of traction. With a greater force, bone is resorbed on *both* sides of the tooth—on the side of distraction as well as that of compression. It is true that distraction inhibits bone formation, but it is also true that, in its osteoclastic response, bone is even more sensitive to compression than to distraction.

Surgeons have been misled by Woolff's law. How often have we reiterated that structure is subservient to function, and that trabecular bone is laid down in a pattern conforming to the lines of weight-bearing? Certainly bone that is functionally inactive becomes porotic, whereas bone that is subjected to an increasing load shows thickening of the trabeculæ; but this again is a defensive response to the *destructive* effects of compression, and moreover it is a defence that is not always successful. In a scoliotic spine the trabeculæ are thickened on the concave side where an excess load is carried, but nevertheless the vertebræ are wedged on that side. In congenital subluxation of the hip joint, lack of pressure of the femoral head allows the floor of the acetabulum to thicken, whereas pressure of the head against the roof of the acetabulum prevents it from growing. The known effect of binding the foot of a Chinese girl, the recognised increase in the degree of genu valgum by the compression force of weight-bearing when deformity is once established, and the proved effects of stapling in arresting the growth of an epiphysis, leave no doubt that epiphyseal growth is arrested by compression, and similarly, in adult bone, alteration of shape in response to postural deformity, or to the pressure of continued splinting, and the sites of bone destruction when deformed joints are submitted to weight-bearing compression, make it clear that bone is resorbed when pressure is increased and laid down when pressure is relieved.

Winston Churchill wrote: "When I have to stand on parade, or even, I regret to say, in church, for half an hour at a time, I have always felt that the erect position is not natural to man, has only been painfully acquired, and is only with fatigue and difficulty maintained." In his humour there was more than an element of physiological truth. Our skeletal framework does not dissolve when we stand erect and submit it to a weight-bearing compression of much more than 20 mm. of mercury only because an elaborate protection from such dissolution has been developed. Whenever two bones are in weight-bearing contact they are separated by plates of cartilage or

fibro-cartilage which are relatively avascular and therefore immune from the destructive effects of compression Thus, in aneurismal erosion of the spine, the intervertebral discs are spared, and when the tibia is eroded by the pressure of a cystic meniscus the plate of articular cartilage survives From these protective plates a trabecular system is built up slowly, in the course of years, so that compression forces are dispersed and distributed widely to the cortical shafts of long bones. Are we to assume that when the recently fractured fragments of a bone are forced and compressed into each other there has been time for the development of such a protective mechanism? I submit that fractures unite *despite* such compression and not because of it, and that the only benefit conferred by lag screws, slotted plates, and transfixion pins with compression, is that fixation is so augmented that shearing and rotation strains are eliminated If living bone surfaces are held rigidly in apposition they unite The benefit of immobility is proved. But the benefit of impaction and forcible compression is far from proved

Moreover, the merit of early weight-bearing in accelerating the union of fractures has not yet been established It would almost appear from the teaching of some surgeons that lower-limb fractures cannot be expected to unite soundly except with the stimulus of weight-bearing; but no one has yet suggested that patients with fractures of the upper limb should walk on their hands and knees, and if weight-bearing compression is essential in fractures of the leg, why is it not essential in fractures of the arm? The only evidence of which I am aware shows the exact opposite far from the union of fractures being accelerated by early weight-bearing, it is delayed A series of 800 fractures of the shafts of the tibia and femur were studied carefully There were 191 strictly comparable fractures of the shaft of the tibia treated by simple manipulation and plaster

RATE OF UNION OF CLOSED FRACTURES OF THE TIBIA, SOME TREATED WITH EARLY WEIGHT-BEARING AND SOME WITHOUT

191 Fractures	United by the 12th Week	United by the 16th Week	United by the 20th Week	United by the 52nd Week
	Per cent	Per cent	Per cent	Per cent
Treated without weight-bearing (73)	50 7	74 0	93 2	100
Treated with early weight-bearing (118)	37 3	69 5	87 3	100
Fractures with residual angulation treated with early weight-bearing (41)	31 7	60 9	80 4	100

When early weight-bearing was not permitted, over 50 per cent of the fractures united soundly within twelve weeks, whereas only 37 per cent. united rapidly when weight-bearing was allowed The average delay caused by early weight-bearing was in the region of four weeks, but delay was still greater if weight-bearing was permitted when the alignment was imperfect. This is no more than might be expected because weight-bearing on an angulated bone causes distraction on the open side of the angle

Summary—The evidence suggests that the cause of non-union is inadequate immobilisation; that metallic internal fixation cannot be relied upon if bone union is unsound, that there is no evidence that forcible "contact compression" aids the healing of fractures except in so far as it augments immobilisation, and that the union of fractures is not accelerated by early weight-bearing I would conclude the chapter, as I began it, by quoting from Hugh Owen Thomas's "Contributions to Surgery and Medicine, 1886-1890".

"*We do not expect union of the bones if motion of the fragments is permitted*"

"*Rest must be physiological as well as mechanical, and uninterrupted so long as the slightest unsoundness exists The mechanical treatment should not trespass upon the physiological*"

"*Pegs, if there be any strain upon them, cannot long maintain their hold when inserted into living matter.*"

"*The practice of jamming the fragments will ere long be found not to be any advantage upon the methods of the past*"

"*I fail to perceive the advantage of compelling them [the patients] early to assume the perpendicular position, which they can maintain but a short time, and so would have frequently to recline upon a couch—a lesser luxury than reclining upon a bed*"

"*If it should be discovered that I have added one atom to the principle, it will give me much more satisfaction than if I should succeed in burdening practice by ingenious means*"

CHAPTER XII

SHOCK, STRESS AND THE ADAPTATION SYNDROME

"The physician who calls himself endocrinologist and confines his interests to such unfortunate members of society as might appear in the side-show of a circus never realises that pneumonia, a broken leg and a bad burn involve important changes in adrenal cortical function"

—FULLER ALBRIGHT, 1943[1]

"A breakdown of the hormonal adaptation mechanism appears to be the most common ultimate cause of death in man."

—HANS SELYE, 1947[2]

The circulatory, metabolic and hormonal disturbances associated with shock are protective physiological reactions, basically no more harmful in their response to injury than fever is harmful in infection. But shock is not a simple clinical entity that can be measured accurately, it is a complex of syndromes in which protective reactions and lethal consequences are distinguished only with difficulty It may be induced by many agents which appear to have little in common, namely. fractures, wounds, hæmorrhages, visceral perforations, surgical interventions, burns and frost-bite, crushing injury of the limbs, prolonged application of a tourniquet without surgical operation or other injury, exposure to heat, cold or lowered barometric pressure, fatigue of muscle and nervous exhaustion, acute infections and intoxications, or any local or general strain for which the organism is not prepared

CLINICAL FEATURES OF SHOCK

Shock may be described as a state of profound physical and mental depression with subnormal body temperature, reduced blood volume, and lowered blood pressure, and with hæmoconcentration, abnormal capillary circulation, impaired oxygenation of tissues, and disturbance of the fluid and electrolyte balance between cells and intercellular spaces The rate of blood corpuscle sedimentation is accelerated and many erythrocytes are clumped into agglutinated masses This "sludging" of blood with blocking of capillaries occurs not only in the region of injury but also in distant viscera which are often the site of embolism by detached clumps of adherent erythrocytes. There may also be circulatory disturbance from traumatic arterial spasm spreading reflexly to proximal parts of the limb, the opposite limb, and even to the renal vessels causing anuria Visceral complications include gastro-intestinal erosion with hæmorrhage causing dark coffee-coloured vomit, and necrosis of the liver—which occurred with particular frequency in former days after severe burns, perhaps in consequence of tannic acid poisoning[3]

[1] Albright, F In Cecil's "Textbook of Medicine," 6th ed Philadelphia Saunders, 1943
[2] Selye, H "Textbook of Endocrinology" 1st ed Montreal Acta Endocrinologia Inc, 1947
[3] Wells, D B, Humphrey, H D, and Coll, J J *New Engl J Med*, 1942, 226, 629



BIOCHEMICAL FEATURES OF SHOCK

The blood chemistry is characterised by reduction of the chloride and sodium content (hypochloræmia and hyponatræmia) from increased leakage of chlorides into the intercellular spaces and sometimes into the pleural, peritoneal or other transudates that may occur, increase in the potassium content (hyperkalæmia) with increased excretion of potassium, rise in blood phosphate, decrease in alkaline phosphatase, and increase in lactic acid The *p*H of the blood shows a tendency towards acidosis Emergency secretion of adrenaline may cause immediate rise in the blood-sugar level with lowering of the liver glycogen reserve, but thereafter the blood-sugar falls to low levels, and when experimental shock is induced in fasting animals there is profound hypoglycæmia

Tissue katabolism with breakdown of body proteins is accelerated There is increase in the total non-protein nitrogen and in the polypeptide, amino-acid, urea and uric acid content of the blood, increase of ketone bodies in the urine, and a negative nitrogen balance The ascorbic acid content of the tissues, especially of the adrenal cortex, is greatly reduced. Hypertrophy of the adrenal glands is associated with involution of the thymus and other lymphatic tissues; and depletion and dissolution of lymphocytes causes lymphopenia despite increase in the total white cell count There is also eosinopenia

These disturbances arise partly from the circulatory changes associated with loss of blood volume, and partly from the general reactions of the organism (alarm reaction), together with nervous and hormonal responses by which defence is established (adaptation syndrome).

GENERAL CIRCULATORY CHANGES IN SHOCK

Many injuries causing shock are associated with external hæmorrhage, but despite loss of volume the blood pressure is usually sustained for a time by vasoconstriction in the skin and splanchnic areas, and sometimes it may even be raised temporarily by the release of adrenaline into the blood stream Nevertheless continued loss of fluid, with increasing reduction of blood volume, lowers the pressure Loss of blood up to two pints is well compensated, but if there is loss of more than two pints—more than one quarter of the total volume—the pressure falls When such loss persists for several hours, anoxæmia of the tissues and increased permeability of the capillaries may give rise to the "death cycle" Pressure in the great veins is so lowered that filling of the heart is decreased and the output is reduced, falling blood pressure is associated with general vasoconstriction, retarded flow from loss of pressure, together with obstructed flow from vasoconstriction, causes capillary stasis and tissue anoxia with progressive loss of fluid to the intercellular spaces, venous return to the heart is thus reduced still more and the cycle is established

Circulatory changes from anæsthesia—Embarrassment of the general circulation may also be aggravated by the anæsthetic that is often needed Any form of inhalational anæsthesia may cause temporary pressor effects from high carbon-dioxide concentration with sudden collapse when the mask

is removed Nitrous oxide, producing cyanosis during administration, may depress the vasomotor centres Excess of pentothal causes respiratory depression Spinal or splanchnic anæsthesia lowers the blood pressure; and regional novocaine anæsthesia is associated with widespread local vasodilatation Open ether is the safest anæsthetic for a patient who is shocked

LOCAL CIRCULATORY CHANGES IN SHOCK

Intravascular agglutination or "sludging" of blood [1-9]—After severe injury, even if there is no external hæmorrhage, the blood volume is reduced by leakage of fluid into intercellular spaces. This can be confirmed by microscopic examination of the capillary circulation In the normal subject a central stream of erythrocytes, separated by a clear space from the walls of the vessel, is seen travelling so fast that individual corpuscles cannot be identified If the tissue is damaged by cold, heat, or mechanical injury, the rate of flow is at once delayed and individual erythroctyes may be identified in the central stream The leucocytes tend to adhere to the capillary walls at the sites of injured endothelium, and diapedesis then occurs with transudation of plasma causing perivascular œdema In many capillaries the corpuscles agglutinate into a clumped mass, stationary except for transmitted pulsation from neighbouring vessels Blocking of some capillaries causes continued change in the direction of flow in others because wherever the circulation is obstructed it is side-tracked through adjacent vessels, sometimes in one direction and sometimes in the other The arterial supply is greater than can be accommodated by the partly blocked circulation and blood is shunted directly into the veins, so that wide areas of circulation may be by-passed At intervals, masses of agglutinated corpuscles are washed free, and examination of far distant tissues—for example, the conjunctiva—shows that countless minute emboli of clumped corpuscles are lodged in many situations where they again cause obstruction of some capillaries, reversed flow in others, and direct arteriovenous shunts in regions that must be by-passed The significance of traumatic intravascular agglutination is not yet understood fully, but it cannot be doubted that clumping of erythrocytes with stasis of capillary circulation is an important cause of fluid-loss into intercellular spaces and that it gives rise to tissue anoxia Extension of such vascular stasis may also explain the phlebothrombosis that often complicates injuries and burns Moreover, embolic blocking of distant capillaries—as, for example, in the kidney causing anuria—may explain many of the complications of shock

[1] Flexner, S "On Thrombi Composed of Agglutinated Red Blood Corpuscles" *J med Res*, 1902, **8**, 316
[2] Fahraeus, R "The Suspension Stability of the Blood" *Acta med Scand*, 1921, **55**, 1
[3] Fahraeus, R "The Suspension Stability of the Blood" *Physiol Rev*, 1929, **9**, 241
[4] Clark, E R, and Clark, E L "Observations on Changes in the Blood Vascular Endothelium in the Living Animal" *Amer J Anat*, 1935, **57**, 385
[5] Knisely, M H, and Bloch, E H "Microscopic Observations of Intravascular Agglutination of Red Cells and consequent Sludging of the Blood in Human Diseases" *Anat Rec*, 1942, **82**, 426
[6] Knisely, M H, Bloch, E H, Eliot, T S, and Warner, L "Sludged Blood" *Science*, 1947, **106**, 431
[7] Brooks, F H, Dragstedt, L R, Warner, L, and Knisely, M H "The Sequence of Circulatory Changes following Severe Thermal Burns" *Anat Rec*, 1948, **100**, 644
[8] Knisely, M H, Eliot, T S, and Bloch, E H "Sludged Blood in Traumatic Shock, Microscopic Observations of the Precipitation and Agglutination of Blood in Crushed Tissues" *Arch Surg*, 1945, **51**, 220
[9] Bigelow, W G, Heimbecker, R O and Harrison R C "Intravascular Agglutination (Sludged Blood), Vascular Stasis and Sedimentation Rate of the Blood in Trauma" *Arch Surg*, 1949, **59**, 667

NERVOUS AND HORMONAL DEFENCES—ALARM REACTION—ADAPTATION SYNDROME[1-5]

Apart from changes in the blood circulation there are many other systemic consequences of injury which have been described as the "alarm reaction." This syndrome may arise from local injury such as a fractured limb or burned skin, or from general injury such as physical exhaustion or exposure to cold. In addition to reduced blood volume with hypotension, hæmoconcentration and accelerated blood sedimentation, there is hypochloræmia, hypoglycæmia, increased tissue katabolism and negative nitrogen balance—the signs of passive damage In quick response to this attack, active resistance is developed by a nervous defence which has long been known to physiologists, and by an even more important hormonal defence which has been recognised only in recent years Manifestations of the alarm reaction then disappear or may even be reversed body temperature rises, the blood pressure returns to normal, the sedimentation rate is slowed; blood sugar reaches hyperglycæmic levels; the chloride and sodium content of the blood is built up, excess of lactic acid disappears, nitrogen balance is restored, and there is enhanced work-performance with increased resistance to injury, cold, fatigue and starvation This is the "stage of resistance."

If the strain continues too long or is too powerful, or if the defences are inadequate to meet it, the "stage of exhaustion" may be reached with reappearance of the signs of the alarm reaction. More often, however, the defences are successful and the organism becomes adapted to the new conditions. The term "adaptation syndrome" covers all phases of non-fatal shock and it embraces the manifestations of passive non-specific damage, intermixed closely with those of active defence

The defence measures are co-ordinated by the hypothalamus and both lobes of the pituitary The afferent pathways, whether nervous or humoral, are not known It may be that nervous tracks between the hypothalamus and pituitary are significant when the stimuli are purely neurogenic, but we do not yet know the route by which messages of distress are carried from an injured limb or burned skin to the anterior lobe of the pituitary which elaborates and releases protective hormones even after transection of the stalk with all its nervous and vascular connections to the hypothalamus

Nervous defence mechanism—In response to stress, impulses from the hypothalamic vegetative centres descend through the splanchnic nerves and induce discharge from the adrenal medulla of adrenergic hormones—adrenaline and *nor*adrenaline There is also direct sympathetic stimulation of smooth muscle with liberation of adrenergic compounds at the nerve-endings in vessel walls Thus vasoconstriction and increased peripheral resistance may be induced in relatively circumscribed areas by direct sympathetic stimulation of the vessels, or more generally by the release of adrenaline into the blood stream. The hypertensive reaction is increased by stimulation of the autonomic nerves of the kidney The renal shunt by which blood is deflected from cortical glomeruli to the juxtamedullary region is opened, and "renal pressor substance" is released so that peripheral vascular resistance is augmented and the blood pressure is raised The nervous

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[2] Selye, H "Textbook of Endocrinology" *Acta Endocrinologica Inc*, *Montreal*, 1949
[3] Laflaquière J "Le choc traumatique" *Société Anonyme de l'Imprimerie* Lyons A. Rey, 1942
[4] Roche, M, Thorn, G W, Hills, A G *New Eng J Med*, 1950, **242**, 307
[5] Selye, H "Stress and the General Adaptation Syndrome" *Brit med J*, 1950, **1**, 1383

system also participates through the hypophyseal stalk and posterior lobe in the regulation of water metabolism, through the hepatic sympathetic nerves in the mobilisation of blood sugar from liver glycogen reserves; and through splenic contraction in inducing changes in the blood count.

Hormonal defence mechanism—The hormonal defence depends upon secretions from the adrenal cortex elaborated under the stimulus and control of the pituitary The importance of adrenal steroid hormones in promoting resistance is demonstrated by the fact that adrenalectomised animals, and patients with Addison's disease, are especially susceptible to shock which may be fatal even after minor operations, but can be prevented if large quantities of the appropriate hormones are given beforehand

Adrenal cortical hormones—There are two groups of adrenal corticoid hormones, namely organic gluco-corticoids such as cortisone or compound E, and compound F, and inorganic mineralo-corticoids such as desoxycorticosterone or D C A In many respects these are antagonists The gluco-corticoids induce resorption of fibrous tissue and inhibit granulomatous reaction, the mineralo-corticoids stimulate the development of fibrous tissue and encourage intracellular protein formation in collagen disease, but each of them shares in promoting general resistance to injury

Control by the pituitary—The defence is initiated by the pituitary—the "conductor of the endocrine orchestra"—which increases its output of corticotrophin (adrenocorticotrophic hormone, A C T H), thus stimulating the adrenals to increased elaboration of such organic hormones as cortisone. Cortisone has been described as "the secret of life" and certainly it is a basic need in defence against every injury that threatens life There is therefore a shunt of activity in the pituitary—unable to produce all its hormones in maximal quantity at the same time—by which elaboration of life-saving corticotrophin is augmented at the expense of reduced output of somatotrophin, the gonadotrophins and thyrotrophin

Gluco-corticoids—Corticotrophin is gluco-corticotrophic The organic gluco-corticoids stimulate the reticulo-endothelial system to increased phagocytosis and antibody formation They induce gluconeogenesis with transformation of non-sugars into carbohydrates, and they are markedly anti-allergenic and anti-histaminic The gluco-corticoids also act upon the thymicolymphatic system and cause involution of the thymus with shrinking of all lymphatic glands, thus being responsible for lymphopenia and eosinopenia Through their effect upon blood proteins they reduce the sedimentation rate

Mineralo-corticoids—Although the response of the pituitary to local or general injury is primarily gluco-corticotrophic, in certain circumstances that are not yet understood clearly it may also be mineralo-corticotrophic. The inorganic mineralo-corticoids influence the sodium, chloride, potassium and water metabolism They increase the output of renal pressor substance and give rise to functional vasoconstriction with raising of the blood pressure.

Ascorbic acid and the corticoids—There is evidence to suggest that the potential of the adrenal cortex in elaborating its hormones is preserved by ascorbic acid. An immediate consequence of the secretion of corticotrophin is lowering of the ascorbic-acid content of the tissues and especially of the adrenal cortex. If animals are deprived of a normal supply of ascorbic acid and are subjected to stress, the stage of exhaustion is reached quickly, whereas if the supply of ascorbic acid is increased adaptation is maintained,

and moreover it is maintained with less hypertrophy of the adrenals[1-4] There is therefore good reason for giving large quantities of this vitamin to patients with severe wounds or major fractures

Estimation of shock by eosinopenia—The capacity of the organism to produce adrenal corticoid hormones is a measure of its resistance, and attempts have therefore been made to assess the cortical reserves before undertaking surgical operations.[5] Such studies have been based upon the eosinopenia that always occurs after secretion of corticotrophin if the adrenal glands are normal. Eosinopenia is to be expected within a few hours of the incidence of any serious injury or major surgical operation and, if it is not observed, adrenal insufficiency should be suspected It must be recognised, however, that even in normal circumstances the eosinophil count varies widely from day to day, and even from hour to hour, so that only major changes in the count are significant.

TREATMENT OF SHOCK

Hitherto the understanding of shock has been difficult because it was not clear why so many different agents caused essentially the same syndrome It was not easy to formulate treatment when the underlying lesion might be a wound with severe hæmorrhage, or a fracture with no external loss of blood a ruptured abdominal viscus, or an overlooked tourniquet left on a limb ; a local injury such as crushing and burning, or a general stress such as exhaustion and exposure , a physical injury such as fracture, or a purely psychological fear such as the witnessing of injury in another Much of the confusion has been resolved by the researches of recent years and still more successful treatment is to be expected when more is known of the hormonal basis of shock Meanwhile, symptomatic treatment must be discussed under the headings : pain and vasovagal collapse ; loss of blood volume ; toxic absorption of the products of tissue injury

Vasovagal collapse, pain and nociceptor stimuli—Vasovagal collapse may arise from a painful stimulus such as that of a sprain or fracture, or from any sensory stimulus with psychological associations of pain such as the collection of donor blood, or the removal of stitches from a wound This is, of course, no more than "fainting" in which there is rapid fall of blood pressure from vasodilatation in all skeletal muscles, apparently through the medium of the sympathetic nervous system, which is aggravated by the upright position It is associated with pallor, heavy breathing, yawning and sweating If the injury is trivial it passes off quickly when the patient lies down or sits with his head between his knees But even in simple vasovagal collapse the blood pressure sometimes falls as low as 50 mm., and this may be at least a contributory factor in the shock of major wounds

In former years the importance of nociceptor stimuli was emphasised to such an extent that abdominal incisions were not made until the tissues had first been infiltrated with novocaine , amputations were not performed without injecting the major nerves before they were divided , spinal

[1] Thérien, M , and Dugal L P "La nécessité de l'acide ascorbique pour l'acclimatation au froid du cobaye" *Rev Canad Biol* , 1947 6, 548
[2] Thérien M , Leblanc J Heroux, O and Dugal, L P "Effets de l'acide ascorbique sur plusieurs variables biologiques normalement affectées par le froid" *Canad J Research*, 1949, 6, 349
[3] Dugal, L P , and Thérien, M 'Influence of Ascorbic Acid on the Adrenal Weight during Exposure to Cold" *Endocrinology*, 1949 44, 420
[4] Thérien, M "Contribution à la physiologie de l'acclimatation au froid' *Laval méd* , 1949, 14, 7
[5] Rud, F "The Eosinophil Count in Health and Disease" Oslo Johan Grundt, Tanum Forlag, 1947



FIG 379





FIG 380

FIG. 381

When there is wide destruction of skin and extensive granulation, external loss of fluid from hæmorrhage and leakage of plasma cause severe shock In this case more than two-thirds of the circumference of the forearm was denuded of skin, most of the ulna was shot away, and there was severe destruction of muscle One pint of plasma was given at the first-aid post and a second pint on admission to hospital, two pints of blood were transfused during the emergency operation



FIG 382



FIG 383

After long treatment including skin grafting, the result was dramatically successful There was no ulna, six inches of the ulnar nerve had been destroyed, and the thumb had been blown off, but function of the hand was excellent and the elbow was mobile and stable



Fig 384



Fig 385

Burns and fractures of the same limb

When there are burns and fractures of the same limb a serious degree of shock must be expected This patient sustained a fracture of the shaft of the femur and a comminuted fracture of the head of the tibia as well as severe burns of the face, arms, hands and legs Transfusion of plasma was started at once.

anæsthesia was advocated in order to block the painful stimuli; and morphine was ordered freely because pain was believed to be harmful in itself Even to-day we cannot exclude the possibility that sensory stimuli may play a part in inducing traumatic shock Nevertheless the emphasis formerly placed on the relief of pain by morphine was unwarranted A patient who is seriously shocked is unaware of pain and it is by no means certain that morphine is useful except in the control of restlessness Spinal anæsthesia has, of course, been abandoned because whatever merit there may have been in blocking painful afferent stimuli, it was more than outweighed by the lowering of blood pressure Experimental evidence confirms that a flow of nociceptor stimuli from the field of injury is not an important cause of circulatory impairment or shock, and although this factor may not perhaps be discounted altogether it is not of fundamental significance.

Shock from hæmorrhage and oligæmia—As long ago as 1876 Blum said that "hæmorrhage is shock, and shock hæmorrhage," and this view has been dominant ever since It might be imagined from reading the literature, even of the last five years, that loss of fluid is the *only* important cause of shock This, of course, is not true, but nevertheless it is well established that prompt restoration of blood volume is a first essential in treatment Loss of blood up to two pints is tolerated because vasoconstriction in the skin and splanchnic areas prevents fall in the arterial pressure, but if more than two pints is lost the blood pressure falls, and if such loss persists for several hours, anoxæmia of the tissues may give rise to the "death cycle" from which recovery is usually impossible

The blood pressure itself is no guide to the severity of oligæmic shock because it may be sustained temporarily by vasoconstriction; it may even be as high as 150–170 mm, while at the same time vasoconstriction, loss of blood volume and tissue anoxia are preparing for a sudden and perhaps fatal collapse from the additional injury of operation or the recurrence of hæmorrhage. Repeated estimation of blood pressure at ten or fifteen minute intervals may offer useful evidence but, as a rule, the indication for transfusion should be based not on the blood pressure, nor on "feeling the pulse," but on the general clinical evidence of probable loss of blood as determined by the severity of the wound.

It is safe to say that if a wound measures "two fists' in volume at least two pints of blood have been lost Such an observation may seem crude when laboratory estimations can be so precise, but in this decision clinical judgment is more important than laboratory tests *If there is doubt, it is better to give a transfusion than to withhold it* A plasma drip should be started at once. At first the rate of transfusion should be fast, though the ultra-rapid methods recently advocated are not recommended If more than two pints of fluid are needed the question of changing over to blood should be considered There is little danger of "over-transfusion" It is true that intravenous saline and glucose saline are not retained in the circulation, and excessive infusion of these fluids may therefore cause pulmonary-œdema. Even with plasma there is some risk when burns are associated with thermal injury of the air passages, or blast injury of the lung, or when there is pulmonary fat embolism But, in general, patients with oligæmic shock cannot be overloaded by the transfusion of two or three pints of plasma [1]

[1] In the treatment of burns it has been estimated that 100 c c of plasma should be given for each 1 per cent of body surface burned, half being given in the first four hours, a quarter in the next six hours and a quarter in the next twelve hours

Shock from circulation of the products of tissue injury—After the first world war it seemed to have been proved by Dale, Cannon, Bayliss, Laidlaw and Richards[1] that injured muscle produced a toxic substance which, when absorbed into the general circulation, caused widespread capillary damage, leakage of plasma into tissue spaces, reduced blood volume and hæmoconcentration. The theory of traumatic toxæmia became associated with the word "histamine," although the original authors claimed no more than that surgical shock *resembled* histamine poisoning. The phrase "histamine shock" was unfortunate because it was soon demonstrated that there was not enough histamine in skeletal muscle to account for the circulatory changes of shock and, moreover, there was no increase of histamine in the blood of injured animals. The hypothesis of toxic shock was discredited.

Recent work by Green, Stoner and Bielschowsky[2-5] has suggested that muscle injury does indeed give rise to a toxic product and that if this is injected into the circulation of a normal animal the signs of shock develop. The toxin arises from nuclear destruction but the nucleotide is decomposed so quickly that it cannot be estimated or even recognised directly in the blood stream. At first the toxin was described as the "muscle-shock factor," and it was shown that this extract of muscles, when injected into a normal animal, gave all the signs and symptoms that develop after removal of a tourniquet from an ischæmic limb. It was then recognised that the extract was adenosine triphosphate.

Experimental work still goes on. For the surgeon who must treat patients with shock after limb injury it can be said only that there is evidence to suggest that harmful toxins may possibly be liberated from destroyed muscle and that injured and ischæmic tissues should be excised quickly. The principles of treatment of patients with severe wounds, compound fractures, or crushing injuries, should be: (i) take the patient immediately to the operating theatre without delay for measures of "resuscitation"; (ii) begin a transfusion of plasma at once; (iii) excise all dead and devitalised muscle as soon as possible.

The overlooked tourniquet—The practical significance of the "muscle-shock factor" arises particularly when a tourniquet has been left inadvertently on a limb for many hours. This accident should not, of course, occur. First-aid workers should not use tourniquets, and in elective operations only pneumatic tourniquets should be used. But supposing that a rubber tube or Esmarch tourniquet has been left on a limb for many hours—the question arises whether it should be removed, with the inevitable consequence of serious shock and uræmia from which the patient may die, or whether the limb should first be amputated above the level of the tourniquet. No case has been reported of survival after the loosening of a tourniquet which had been in position for nine hours or more. Probably six hours should be agreed as the determining time. *If an overlooked tourniquet has been in position for six hours or more it should not be released; the limb should first be amputated at a more proximal level.*

[1] Dale, Cannon, Bayliss, Laidlaw and Richards. Medical Research Council of Great Britain. Report of special investigation committee on surgical shock and allied conditions. Nos. 1 to 7, 1919. Report No. 25.
[2] Green, H. N., and Bielschowsky, M. "Shock-producing Factor from Striated Muscles." *Lancet*, 1943, 147.
[3] Stoner, H. B., and Green, H. N. "Adenosine-compounds and Phosphates in the Blood of Shocked Rabbits." *J. Path. Bact.*, 1944, 56, 343.
[4] Green, H. N., Stoner, H. B., and Bielschowsky, M. "The Effect of Trauma on the Pentose Content of the Plasma in Animals." *J. Path. Bact.*, 1949, 61, 1, 101.
[5] Green, H. N., and Stoner, H. B. "Present Status of Adenine Nucleotides in Bodily Response to Injury." *Brit. med. J.*, 1950, 1, 805.

CHAPTER XIII

OPEN FRACTURES AND JOINT WOUNDS

Nearly two centuries ago Percivall Pott was thrown from his horse in the Old Kent Road and sustained an open fracture of the shaft of the tibia At that time the penalty of wound infection was amputation of the limb or death of the patient and Pott, who knew it would allow no one to move him Lying on the road in the depths of midwinter he sent to Westminster for two chairmen with their poles, meanwhile purchasing a backyard door to which the poles were nailed On this improvised stretcher he was carried over London Bridge to Watling Street near St Paul's Not only did he survive the injury but he escaped amputation of the limb. It was a triumph of first-aid treatment.



FIG 386

Percivall Pott (1714-88)

From a portrait in the Royal College of Surgeons of England, reproduced in the *Journal of Bone and Joint Surgery*, 1949

Protection from further injury is a basic principle in the treatment of open fractures There are, in fact, eight principles of treatment which may be considered in sequence of time under the headings

1 First-aid protection ,
2 Treatment of wound shock ;
3 Excision of the wound ,
4 Chemotherapeutic control of infection ,
5 Restoration of skin cover by delayed primary suture or skin grafting ,
6 Final control of the infection by sequestrectomy ,
7 Replacement of unstable scars by whole-thickness skin grafting ,
8 Late reconstructive surgery.

FIRST-AID TREATMENT

Splint them where they lie—If a splint is available it should be applied before the patient is moved In ideal conditions a Thomas' splint should be fitted before the boot is taken off or the trousers are cut, so that traction can be applied from the boot itself by means of a skewer through the heel or a clip on the waist (Fig 387) Alternatively, first-aid splints may be improvised from pieces of wood, strips of metal or even layers of folded newspaper The patient should be placed on a stretcher and not be moved from it until expert treatment is available

Emergency tourniquets are weapons that kill—Hæmorrhage should be arrested by pressure applied directly over the site of bleeding A thick pad bandaged firmly over the wound will stop nearly all bleeding First-aid workers should not be given tourniquets ; the temptation to use them is

too great A tourniquet that is not applied tightly enough increases the hæmorrhage and the patient may then bleed to death a tourniquet that is applied too tightly kills the patient by shock. Gangrene is the least of the complications that arise from the use of this dangerous instrument—devised to control hæmorrhage from which, in point of fact, the victims

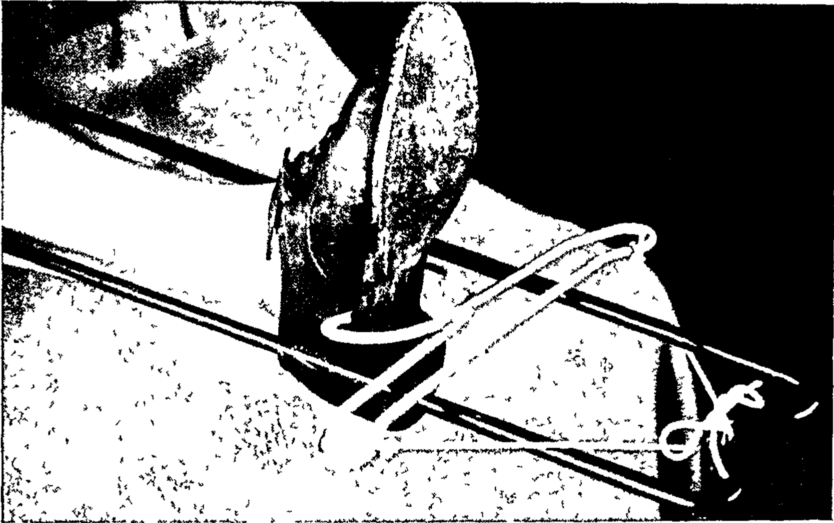


Fig 387

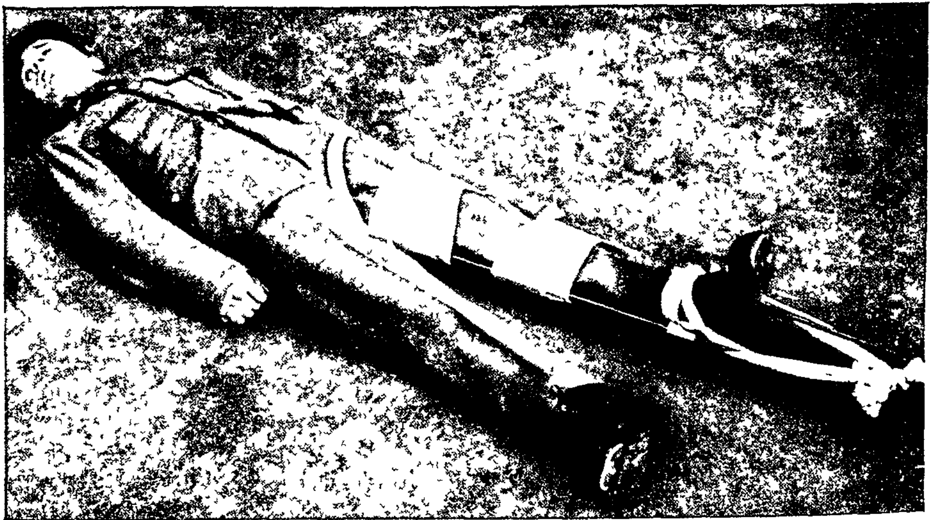


Fig 388

The Thomas' splint is the ideal first-aid splint for any lower-limb fracture Light traction may be maintained from a clip on the waist of the shoe (Fig 387), or from a clove-hitch round the ankle (Fig 388) The clove-hitch must not be used for more than an hour or two because it will cause sloughing round the ankle

of injury seldom die If the patient is left alone, hæmorrhage from a completely severed artery is soon controlled and stopped by reflex vasospasm. More lives have been lost by the use of tourniquets than ever were saved

The patient should be protected from cold by blankets placed under as well as over him ; and the foot-end of the stretcher should usually be raised. Water, with the addition of half a teaspoonful of salt to the pint, may be given freely and repeatedly It is sometimes wise to relieve pain and

restlessness by morphine, but here again there are natural defences The more grave the injury, and the more severe the shock, the more complete is the analgesia, shock itself is an anæsthetic and seldom needs to be supplemented by drugs

2 TREATMENT OF WOUND SHOCK

Wound shock was discussed in the last chapter but the conclusions may be summarised (1) The essential basis of shock is hormonal and the measure of resistance of an organism to local injury, as to any other strain, is represented by its capacity to produce adrenal corticoid hormones under the control of the pituitary The adrenal cortex is sustained in its activity by vitamins, particularly ascorbic acid (2) Afferent nociceptor stimuli and the central effects of pain, or even the psychological fear of expected pain, represent only one form of strain to which adaptation can be established if the hormonal response is adequate, and the control of pain by morphine, or the blocking of afferent stimuli from injured areas by novocain infiltration or spinal anæsthesia, is no longer regarded as an essential basis of the treatment of shock. (3) Devitalised tissues may produce toxic products which cause circulatory collapse when released into the blood stream and, although proof of such toxic products is still lacking, prompt excision of dead and dying tissue may assist in preventing the development of shock. (4) Leakage of plasma into intercellular spaces, which occurs even when there is no open wound, aggravates loss of fluid from external hæmorrhage or drainage of plasma from burned and denuded areas of skin, and restoration of blood volume by the transfusion of plasma or whole-blood is a first essential in the treatment of wounds and open fractures Blood pressure readings are often misleading because even when there has been considerable loss of blood the pressure may be sustained temporarily by vasoconstriction The patient should be taken to the operating theatre as quickly as possible in order that transfusion of plasma or blood may be started at once and the wound be excised without delay

3 EMERGENCY TREATMENT OF THE WOUND

An essential measure in the emergency treatment of wounds is the excision of dead and dying tissue It has already been shown that this may be important in preventing shock, it is certainly important in preventing infection Sir Alexander Fleming, discoverer of penicillin, said "The greatest of all antiseptics is living tissue" Carbolic acid and other powerful antiseptics are useless because they can destroy only bacteria that lie on the surface and, moreover, they also destroy cellular tissues It is upon the vitality of tissues that the real defence against infection depends

> "*Behold a sower went forth to sow And it came to pass as he sowed, some fell on stony ground, where it had not much earth; and when the sun was up, because it had no root, it withered away And other fell on good ground, and sprang up and increased, some thirty, some sixty and some an hundred* ."

A wound with necrotic debris in its depth, and devitalised tissue in its walls, is good ground upon which the seed of bacteria may spring up and





Fig. 389

Compound fracture of the shafts of both bones of the forearm treated by prompt excision of the wound

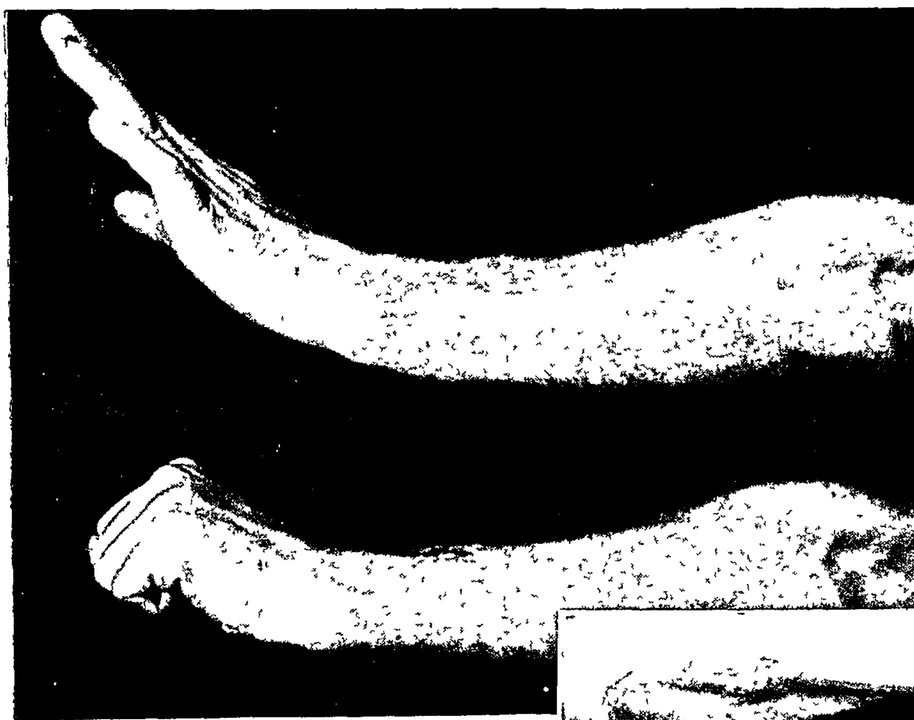




Fig 390

Early operation prevented infection There is normal finger movement and practically no disability The fractures are united in good position

increase, whereas a wound from which all dying tissue has been excised, the walls of which are living and pulsating with a rich supply of blood, is ground upon which bacteria cannot survive. The first essential of wound treatment is excision of dead and dying tissue, and not one of the recent developments of chemotherapy can alter this principle. No matter how effective penicillin, streptomycin, aureomycin or any other antibiotics may be, they are useful only when they can reach the pathogenic bacteria. If micro-organisms are locked away in the depths of a mass of dead tissue that has no circulation, antibiotics cannot reach them. Moreover, chemotherapeutic agents are useful only so long as the bacteria are sensitive to them, and there is evidence to suggest that as new antibiotics are discovered new strains of insensitive bacteria are bred. We must not be lulled into false security by the dramatic progress of chemotherapy. The first principle of wound treatment remains—excise all dead and dying tissue.

Arrangements for operation—*Danger of sudden movement of the patient*—From the time that the patient reaches hospital, is admitted to the ward, and is transferred to the operating theatre, care should be taken to avoid sudden lifting or turning movements. The circulation of a patient with shock is precarious to such a degree that any sudden movement or lifting is tolerated badly. He should be brought to the operating theatre in his bed, lifted only to the operating table, and even then lifted with great care. He should not be turned over unless it is essential. It is sometimes wise to arrange an X-ray examination, but when this is needed it should be done with a portable apparatus while the patient lies on the operating table.

Blood transfusion—The sooner the blood volume is restored the better. A plasma drip should be started at once and be continued throughout the operation. In severe states of shock the rate of transfusion may at first be increased. Very rapid transfusion has recently been suggested, the plasma or blood being forced out of the transfusion bottle by means of oxygen under pressure, thus reaching a rate of even 250 ml. per minute.[1] Such speed of restoration of blood volume has certain advantages, but it increases the risk of air embolism and of overloading the circulation.[2] In this country, rates of 100 ml. per second have seldom been exceeded, and it is usually enough to use the simple and safe method of a wide-bored needle (24/10 instead of 15/10 gauge) with the aid of gravity by raising the level of the transfusion bottle. Sometimes transfusion may be given simultaneously into two different veins. If it is decided to transfuse blood, very special precautions must be taken to avoid a mismatched transfusion, particularly because in the anæsthetised patient there are no symptoms of such mismatching and the signs resemble those of shock. In elective operations this danger is excluded by careful primary matching of the blood, but even in emergency operations the danger is so great that blood should not be transfused to an anæsthetised patient unless similar precautions have been taken.[3]

[1] Pierce, V. K., Robbins, G. F., and Brunschwig, A. *Surg. Gynec. Obstet.*, 1949, **89**, 442.
[2] Drummond, R. J. *Brit. med. J.*, 1943, **2**, 319.
[3] **Mismatched blood transfusion**—*Late hæmolytic reaction*—For the first few days after massive blood transfusion the possibility of hæmolysis from mismatching of blood must be borne in mind. The symptoms are secondary collapse with a cold clammy skin, dyspnœa and vomiting, followed by transient jaundice, increasing oliguria and finally anuria from blocking of the renal tubules with acid hæmatin from the foreign hæmoglobin. The urine must be alkalinised; a mixture of potassium citrate (gr. xxx), sodium bicarbonate (gr. xxx), syrup (m xxx), water to 1½ oz. is given orally four or five times in twenty-four hours. If anuria persists for forty-eight hours, saline with 3 per cent. potassium citrate and 3 per cent. sodium bicarbonate should be given by intravenous drip; the saline transfusion should be discontinued as soon as possible because there is danger of pulmonary œdema and cardiac failure.



FIG. 391

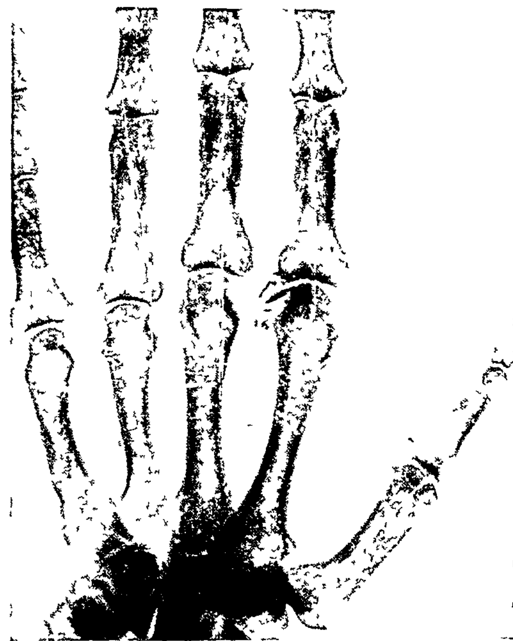


FIG 392

Foreign bodies in wounds

Figure 391—Piece of tubular glass in the palm   Figure 392—Fragments of wood in the base of the index finger, the shadows are dense because they are pieces of plaster-lath from the roof of a bombed house



FIG. 393



FIG 394

Typical foreign bodies of war wounds

Figure 393—Mills bomb wound the foreign body shown in the inset is the cap of the bomb   Figure 394—Incendiary bullet wound (the plug of phosphorus is seen above the nose of the bullet); after incision of the wound and entry of air, combustion of the phosphorus causes "smoking" Symptoms of phosphorus poisoning have been recorded in such wounds. (*A J Blaxland and J W Buttery*, "*Brit med J*," 1942, 2, 664 and 767.)

Anæsthetic—Open ether is the anæsthetic of choice. In a patient who is shocked, induction with pentothal is unnecessary and unwise Gas and oxygen with ether may be used but, since the reduced oxygenation of tissues which is characteristic of wound-shock is aggravated by cyanosis and asphyxia, gas should be given only by an expert anæsthetist Spinal anæsthesia should never be used

Tourniquet—A tourniquet should not be applied because if the field is bloodless it is difficult to distinguish living vascular tissue, which need not be excised, from dead avascular tissue, which must be excised Moreover, the blood supply should not be reduced even for the thirty or forty minutes of operation The tissues are already devitalised, and if they are deprived of blood for half an hour or more bacterial growth is encouraged and infection may be established

EXCISION OF THE WOUND

The wound should be covered with a pad of sterile gauze while all surrounding skin is cleaned with ether or ether-soap and a detergent such as "cetavlon" or "phisoderm," or an antiseptic such as iodine A very narrow strip of the bruised skin margins should be excised, it is usually sufficient to remove one or two millimetres Wide excision of skin is seldom needed and is to be avoided whenever possible.

Division of fascia—The wound should be extended in the long axis of the limb, both proximally and distally, so that all injured deep tissues can be exposed easily without heavy retraction or burrowing in undermined pockets In most compound fractures of civilian life there is only one wound, but in compound fractures from explosives there is usually a wound of entry and a larger wound of exit, both of which must be excised through incisions on opposite sides of the limb All intermuscular spaces should be opened and blood clot evacuated, the deep fascia being divided freely so that swelling of muscles can occur without strangulation and ischæmia

Excision of crushed muscle—The most important part of the operation is the excision of crushed and dead muscle Working systematically from superficial to deep layers the wound should be explored and all damaged tissue excised cleanly with curved scissors Changes in the appearance and colour of the muscle, loss of contractility on mechanical stimulation, and failure to bleed all demand wide excision of this most vulnerable tissue, especially when gas gangrene is suspected In most parts of the limbs, particularly the thigh, there is so great a reserve of power that wide areas of muscle can be excised without functional incapacity

Removal of foreign bodies—Fragments of clothing wood, grass or mud, and, in the case of war wounds, shell fragments or bullets, should be removed On the other hand, when the tissues are peppered with minute foreign bodies, as for example after shot wounds, the tiny splinters cause minimal devitalisation and quick healing occurs despite the foreign bodies (Fig. 395) In many war wounds even larger metallic foreign bodies may be scattered so diffusely that it would be impossible to remove them all (Fig. 396) Foreign bodies should be lifted from the cavity and walls of the wound, but there need be no wide exploration beyond its limits.

Conservative excision of fragmented bone—Excision of fragmented bone should be as conservative as possible A bone fragment that is wholly



Fig 395

Lead shot wound sustained during clay-pigeon shooting Excision of all foreign bodies is obviously impossible.



Fig 396

Wire, fragments of aluminium and other foreign bodies widely scattered



Fig 397

Shell wound causing multiple fractures of the foot and widespread peppering of the tissues with small metal fragments

denuded of all soft-tissue attachment should be removed carefully, but no bone with adherent soft tissue should be excised, no matter how flimsy the attachment or precarious the blood supply There must of course be no question of dissecting the bone or roughly twisting it from the wound Excision of all segments of the shaft of a long bone has the particular danger of causing collapse of the periosteal tube and consequent established non-union of the fracture (Figs 77-78, p 34)

Burying foreign bodies—Since one of the main purposes of operation is the removal of foreign bodies, there can be no justification for burying other foreign bodies such as wire, screws or plates Even sterile catgut is a foreign body which causes sero-fibrinous exudation and increases the danger of infection, and the less catgut that is buried the better. Hæmostasis can usually be achieved by applying artery forceps to each bleeding point and twisting them at the end of the operation. Only large vessels need ligation with thread or fine catgut In no circumstances should fascia, muscles, periosteum or any other deep layer of the wound be sutured Swelling and expansion of tissues must be permitted without risk of tension, and, far from the fascial layers being sutured, they should be divided freely and left wide open

Severed nerves and tendons—Severed nerves and tendons should not be sutured at the emergency operation except occasionally when a cleanly incised wound will almost certainly heal by first intention. It is not even advisable to explore wounds widely with the object of determining whether or not nerves are divided Little advantage is gained by primary suture of tendons or nerves No harm arises from deferring repair until a second-stage operation after the wound is healed soundly The emergency operation should be concentrated on the steps needed to secure prompt healing without infection

Suture of the skin—Although it is true that the deeper layers of a wound should seldom be sutured, there are circumstances in which the skin should be sutured In sucking wounds of the chest and penetrating wounds of the brain immediate skin suture is imperative, and in wounds of a joint it is often advisable In other wounds, skin suture is never imperative and never advisable unless it is almost certain that they will heal without infection Many cleanly incised wounds heal by first intention ; but when there is extensive destruction and crushing of muscle the balance between prompt healing and infection is precarious If skin is sutured the tension of accumulating exudates may impair the circulation and so cause infection If there is doubt it is better not to suture the skin but to lay a few strips of gauze lightly between the walls and prepare for secondary suture after a few days The only advantage of primary skin suture is the slightly improved cosmetic appearance of the scar, and against this must be balanced the serious possibility of wound infection Is gambling justified when the stakes are a neat scar against an amputated limb ? The answer cannot be in doubt Only in the cleanly incised and relatively uncontaminated wounds of normal civil life is immediate skin suture safe In penetrating crushing wounds, and in the wounds of war, immediate suture is seldom safe If it seems certain that a wound will heal without infection, cover it with gauze and let it heal ; if it is not certain that it will heal without infection, separate the walls with a light gauze dressing. in neither case take the unnecessary

risk of suturing the skin At all costs do not half suture the skin; do not lay in strips of rubber which admit infection more certainly than they drain it (Fig 398)

These observations regarding the dangers of primary skin suture apply no less forcibly now that chemotherapy is available Even massive doses of penicillin, aureomycin and other antibiotics are inadequate as compared with the protection of surgical excision and relief of tension. Moreover, there can be no support for the view that when chemotherapy is available the operation of wound excision is no longer urgent and that it can be performed at leisure. No antibiotic can reach the depths of a mass of dead muscle that has no circulation, the action of sulphonamides is inhibited

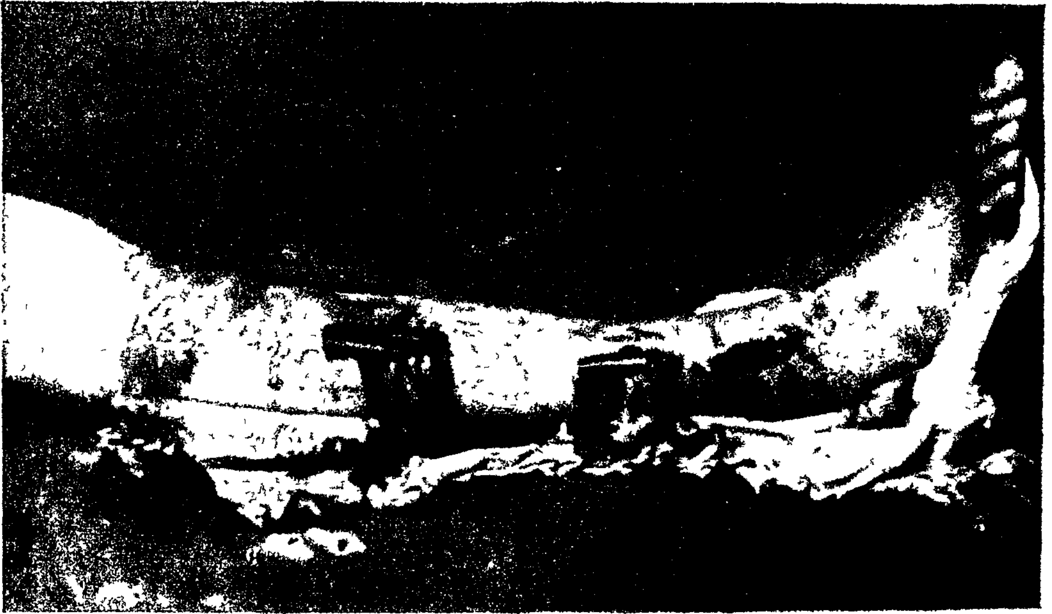


Fig 398

Wrong way of treating an infected wound

Multiple stab incisions are useless, and rubber drains are dangerous, particularly as in this case if the limb is then enclosed in plaster The abscess cavity should be laid open so freely that rubber drains cannot be used because they would fall out

by dead material and by pus fluid, and many bacteria are not sensitive to penicillin Chemotherapy is a most valuable adjunctive treatment, but it cannot take the place of wound excision and it cannot alter the principles upon which that operation is based.

Immobilisation of the limb—John Hilton, Hugh Owen Thomas and others taught that inflamed tissue is capable of dealing with its enemies unaided if kept completely at rest The purpose of immobilisation is not only to avoid the injury of movement but also to reduce the flow of lymph and thus localise infection If a limb is immobilised, the flow of lymph almost ceases It is muscle contraction and particularly joint movement that squeeze infection into the lymphatic meshwork. The joint above and below the wound should therefore be immobilised This can usually be done by means of a plaster slab applied to one side of the limb and bandaged lightly into position It is seldom necessary or advisable to apply a complete

encircling plaster cast at this early stage In fractures of the thigh and leg the support of a Thomas' splint with light traction should also be used.

Elevation of the limb—As soon as the patient is back in bed, and for several days after operation, the limb should be elevated and kept on a plane higher than the heart The injured region should never be dependent even for short intervals Gravitational œdema increases tension within the wound, and it delays healing and promotes stiffness of the joints. Even joints that are susceptible to stiffness, as for example the joints of the



Fig 399



Fig 400

Same case as Figure 398 When the abscess cavity is laid open freely throughout its length (Fig 399) healing occurs rapidly Figure 400 shows the condition six weeks after operation

fingers, retain their mobility despite immobilisation in plaster for a few weeks provided that constant elevation is maintained so that the tissues are never soaked in sero-fibrinous exudates

Excision of Bullet Wounds

In wounds inflicted by hard bullets of high velocity it is often sufficient to excise the small wound of entry and the equally small wound of exit, because the track is cleanly drilled, the destruction of soft tissues is minimal, and foreign bodies are seldom left in the wound An Air Force pilot was shot in the palm of the hand and the bullet, having travelled almost the whole length of the limb, lay beneath the triceps muscle in the upper arm. Not one muscle, tendon, artery, nerve or bone was severed The bullet was removed and the wound in the palm excised, but the track in the forearm



Fig 401

Compound fracture of patella with wide opening of knee joint
The wound was excised Only two catgut sutures were buried



Fig 402



Fig 403



Fig. 404

Excision of joint wounds

Same case as Figure 401. There has been no infection Knee movement is satisfactory, there is normal power in the quadriceps, and the fracture is united firmly.

and upper arm was not exposed. Healing was by first intention and, despite deep hæmorrhage in the forearm and causalgia of the hand from median neuritis, graduated finger exercise prevented stiffness or contracture, and recovery was complete within eight weeks.

Excision of Wounds of Joints

The treatment of penetrating wounds of joints is essentially the same as that of open fractures; the wound must be excised, the joint immobilised, and chemotherapy instituted.[1, 2] If excision is performed within a few hours there is often healing without infection, and good or even normal movement is regained despite the immobilisation. During the 1914-18 war, Willems advocated early mobilisation of acutely infected joints in order to prevent ankylosis and preserve movement, but as Hugh Owen Thomas had already written: "It is as reasonable to attempt to cure a fever patient by kicking him out of bed as to benefit joint disease by wriggling at the articulation." If the principle of continued and uninterrupted rest is accepted, even gravely injured and grossly contaminated joints, treated promptly by surgical intervention and chemotherapy, are much less likely to become infected than was believed possible in former years. Wounds of ligament, synovium and articular cartilage are, in fact, more resistant to infection than wounds of muscle and cellular tissue, and primary skin suture is often safe.

Wounds of the knee joint—After excising devitalised tissues and removing foreign bodies a few interrupted sutures should be inserted in the capsule, and if the patella has been fractured the quadriceps expansion should be approximated by suture (Figs 401-404). Accurate reconstruction of the patella, or excision of the bone with repair of the quadriceps tendon, should usually be deferred and, if need be, performed at a second operation after the wound is healed. If the wound is excised within a few hours of injury the skin may also be sutured. The limb should not be immobilised in a complete plaster cast. Enclosure in plaster is safe only when wounds are left widely open; and moreover first-intention healing is promoted by dryness of the skin and hindered by the moisture of serous discharges and sodden dressings, which encourage the surface growth of bacteria. It is true that constitutional signs and the temperature and pulse charts, are the most reliable evidences of general infection, but it should also be possible to inspect the wound easily and to remove the skin stitches if local infection develops. A Thomas' splint should therefore be used with light traction.

When many hours have elapsed since the time of wounding there is a greater danger of infection of extracapsular cellular tissues but still a likelihood that the joint itself may remain free from primary infection. The greatest peril is the spread of infection to the joint from extracapsular tissues. In these circumstances the synovial and capsular layers may still be sutured, but the skin should be left open with a gauze dressing laid lightly between its margins. If there is no infection, delayed primary skin suture may be undertaken on the third or fourth day. Even when there is low-grade infection so that skin suture is not possible, and the light gauze pack must be retained until the wound heals by granulation, excellent function

[1] Ellis, V H "Pyogenic Affections of Joints and Bones" In "Modern Trends in Orthopædics," edited by H Platt London Butterworth & Co (Publishers) Ltd 1950

[2] Thompson, J E, and Berry, F B "Penetrating Wounds of Major Joints" *Ann Surg*, 1947, 126, 947.



Fig 405



Fig 406



Fig 407

Excision of penetrating bullet wound of joint

Bullet wound penetrating the knee joint with destruction of femoral condyle and tibial tuberosity The foreign body was removed and the wound lightly packed with gauze No primary or secondary suture was performed Nevertheless 120° of knee movement was regained with almost normal power and stability, and full duty was resumed within eight months with an A1 category

is often regained (Figs 405-407) Only if acute purulent arthritis becomes established is ankylosis of the joint inevitable The joint must then be laid open freely through a parapatellar incision extending to the upper limit of the suprapatellar pouch, together with incisions into the postero-medial and postero-lateral compartments Immobilisation in a plaster spica should be continued until healing is complete and ankylosis is sound

Wounds of the ankle joint—The ankle joint should be exposed through an antero-lateral incision in front of the fibula, and if necessary through a posterior incision beside the tendo-Achillis The talus should not be removed unless it has been dislocated and deprived of blood so that it is a sequestrum. Function after astragalectomy is very poor, whereas the results of fusion of the ankle joint, or even of both ankle and subastragaloid joints, are much better

Wounds of the hip joint—The postero-lateral approach of Kocher and Langenbeck is the best for most wounds and infections of the hip joint. The incision lies between the gluteus maximus behind and the gluteus medius and minimus in front and if the insertions of these muscles are partly detached from the greater trochanter free exposure of the joint is gained [1] Excision of the head and neck of the femur has been advocated in the treatment of penetrating wounds, but it is to be avoided at all costs unless infection is so grave that life is imperilled Such wide excision certainly offers very free drainage of the joint, but it causes serious shortening and an unstable painful hip, whereas ankylosis of the joint in the optimal position causes minimal disability A frame or windowed plaster spica may be used with the limb in neutral rotation and at right angles to the pelvis—neither abducted nor adducted

Wounds of the shoulder joint—In the shoulder joint, as in the hip, excision of the head of the bone is a destructive and mutilating procedure which is not justified except very occasionally as a life-saving procedure There is no more difficult problem than that of stabilising a shoulder joint which has been made flail and almost useless by removal of the upper end of the humerus Excision of the wound and drainage of the joint can almost always be achieved without resection of bone The limb should be immobilised on an abduction frame in 60 degrees of abduction and in forward flexion and external rotation

Wounds of the elbow joint—In excising wounds of the elbow joint special care must be taken to avoid injury to the ulnar nerve on the medial side and the radial nerve on the lateral side A vertical posterior incision close to the olecranon is safe The consequences of free excision of bone are less disastrous than in the shoulder or hip joints because arthroplasty of the elbow is nearly always successful Nevertheless if too much bone is excised at the primary operation the false joint will be unstable or even flail, and arthrodesis is then necessary In the case shown in Figures 408-410 it is perhaps remarkable that the general-practitioner surgeon who first saw the patient in a remote cottage hospital did not amputate the limb on sight Wide areas of the limb were denuded of skin , the brachialis and triceps muscles were blown out , the radial and ulnar nerves were injured , the lower end of the humerus was shattered and the joint was destroyed But the brachial artery was intact and the surgeon rightly saved the limb So much bone had been destroyed by the injury, or was excised at operation, that the false joint

[1] Gibson, A "Posterior Exposure of the Hip Joint" *J Bone Joint Surg*, 1950, 32-B, 183



FIG 408

Shell wound in which wide areas of skin were destroyed. The brachialis and triceps muscles were blown out, the radial and ulnar nerves were injured, and the lower end of the humerus was shattered The brachial artery was almost the only intact structure—but the *one* needed to justify saving the limb



FIG. 409



FIG 410

Same case as Figure 408 After excision of the wound, free skin-grafting, and radio-humeral arthrodesis (with preservation of radio-ulnar movement), excellent function was regained and the patient fought again as a pilot despite the stiffness of the elbow.

was functionally useless and had to be arthrodesed Nevertheless radio-ulnar movement was preserved, the nerve injuries recovered, and the patient regained a limb so useful that he resumed duty as a pilot and fought in France

Excision of Head Wounds

Wounds of the scalp, especially war wounds, are potentially serious because the wound is often more extensive than first inspection would suggest, and if there is an associated injury to underlying bone and brain the predisposition to infection is strong Whenever possible these cases should be treated by experienced neuro-surgeons in fully equipped theatres Early excision of the wound is of vital importance, particularly when there is evidence of underlying craniocerebral injury Cruciate enlargement of the wound is inadequate, there must be wide exposure by means of large scalp flaps Blood clot and extruded brain matter are removed by means of a sucker Fragmented or devitalised bone is freely removed Unlike compound fractures of the limbs where wide excision of bone has unfortunate sequelæ, defects in the skull from radical treatment can be repaired with relative ease The dangerous sequel is not non-union but infection There must be no hesitation, therefore, in removing bone which is contaminated or devitalised The dura is opened Damaged brain tissue, clot and bone fragments are sucked out of the cavity Hæmostasis is secured by clips or diathermy coagulation The dura is then sutured The exposed area must be covered by sound scalp which has been carefully sutured If scalp tissue has been destroyed in the region of the wound, flaps should be swung into position even at the cost of denuding other areas where periosteum and bone are uninjured, these areas heal by granulation and can be skin-grafted at a later date.

Excision of Chest Wounds

A penetrating wound of the chest is a serious emergency Two types of wound are to be differentiated (1) valvular wounds which allow entry of air into the pleural cavity during inspiration but prevent its exit during expiration, thus causing a tension pneumothorax ; (2) sucking wounds which allow free entry of air during inspiration, and free exit during expiration, thus causing an open pneumothorax In each case excision of the wound and air-tight closure of deep tissues and skin give rapid relief Deeply placed silkworm sutures, including skin, muscle and deep fascia, should be used A moist dressing is applied or a dressing of gauze impregnated with paraffin emulsion If there is a tension pneumothorax, a short wide-bore needle is inserted into the pleura through the second intercostal space, two inches from the margin of the sternum If necessary the needle is left in position and connected with a water-seal bottle

CHEMOTHERAPEUTIC CONTROL OF INFECTION

The history of antibiosis dates back to the beginnings of folk medicine Few knew how extensive were the groping efforts of many pioneers throughout a half century, in their attack upon one micro-organism after another, until they read the Lister Lecture delivered by Howard Florey in 1945 and the reader is referred to the latest publication by Florey and his colleagues which gives a detailed history of these studies together with the characteristics



Fig 411
Lord Lister (1827-1912)
Portrait in the Royal College of Surgeons, reproduced in the *Journal of Bone and Joint Surgery*, 1948

of all known antibiotics produced by fungi, actinomycetes, bacteria and higher plants—a review that is almost bewildering in its completeness [1]

Notable success in attack upon a specific infective organism was achieved by Ehrlich in 1904 when he introduced salvarsan Thirty years later it was found that the sulphonamide group of drugs had bacteriostatic action on the streptococcus pyogenes, pneumococcus, meningococcus and gonococcus by interference with their metabolism so that they became susceptible to the phagocytic cells of the host. But it was not until the culture plates of Sir Alexander Fleming were studied in his laboratories in London, and penicillin was discovered that the real significance and immense possibilities of antibiotic treatment were recognised

Penicillin, the extract of a mould of the genus *Penicillium*, was proved to be bactericidal to the staphylococcus, streptococcus pyogenes pneumococcus, gonococcus, meningococcus, clostridia of the gas gangrene group, and certain spirochætes including the treponema pallidum Some pathogenic organisms were quite insensitive to penicillin—for example, the large coli-typhoid group, enterococci bacillus proteus and pyocyaneus, and the tubercle bacillus Since then the whole plant kingdom has been the subject of world-wide search for sources of new antimicrobic agents, and preparations such as streptomycin, aureomycin choloromycetin and terramycin have appeared These are but a few of the host of substances that are now being studied in laboratories and applied in clinical treatment, and during the next few years it will be difficult indeed for any standard textbook to keep pace with the developments of antibiosis, and to appraise the benefits and dangers of the many specific agents that will be introduced A new era of treatment has been introduced by a great British discovery



Fig 412
Sir Alexander Fleming, who discovered penicillin, in his London laboratory

It was first believed that in controlling infection by penicillin the ideal administration was a continuous intravenous drip which maintained the blood concentration at a constant and optimal level Although this unpleasant treatment was theoretically sound, it often failed by reason of thrombosis of the recipient vein, and was replaced

[1] Florey, H W, Heatley, N G, Jennings, M A, Sanders, A G, Abraham, E Q, and Florey, M E "Antibiotics" 3 vols London Oxford University Press, 1949

quickly by three-hourly intramuscular injections repeated throughout the day and night. It is now known that larger doses injected at less frequent intervals are no less effective. The standard treatment to-day is to give from one-quarter to half a million units of penicillin twice in twenty-four hours. The dried crystalline salt, made up in small bottles which should be kept in a cool place, is dissolved in 1 c c of sterile water and injected intramuscularly. Penicillin in procaine may be used to relieve local pain, and penicillin suspended in oil is sometimes recommended in order to give a slower rate of absorption.

Penicillin in emergency treatment—In the treatment of severe wounds and open fractures one million units of penicillin should be injected forthwith, together with one-quarter million units twice daily thereafter for three or four days. This is prophylactic treatment, to be instituted at once even before there is time for bacteriological investigation. When infection is established laboratory tests are needed to determine the pathogenic bacteria in the wound and their sensitivity to various antibiotics. If the bacteria are penicillin-sensitive the dosage of penicillin should be increased to one million units daily, and continued until all signs of infection have disappeared; but if they are penicillin-resistant one of the other antibiotics, to which they are sensitive, should be used.

Freedom of penicillin from toxic reaction—Penicillin is still unique in its freedom from toxic reaction despite high dosage. Minor toxic effects have been observed—penicillin fever, dermatitis, arthralgia, glossitis and urticaria with angioneurotic œdema, sometimes necessitating the injection of antihistamine drugs such as "antisan" or "phenergan", but these reactions usually arise from impurities in the preparation, and the crystalline sodium salt is now prepared in a form so pure that even long-continued and intensified penicillin treatment is safe. In one case, a child with many years' history of chronic osteomyelitis of the pelvis and femur, with ankylosis of the hip joint in right-angled deformity, severe secondary lordosis, and many sinuses in the abdomen, perineum, trochanteric region and thigh, whose infection had failed to respond to penicillin in small dosage, was given one million units each day for eight weeks, by which time the infection was healed and the sinuses were dry. The deformity was then corrected by trochanteric osteotomy performed through the site of very recently healed sinuses. After operation, one million units of penicillin were given daily for ten weeks. This patient, after enduring for many years the miseries of crippling and deformity, and the twice-daily dressing of many sinuses, received more than two-hundred million units of penicillin, and so was able to undergo an operation which until recent years would have been impossible but which has now reduced her disability to negligible proportions. Even in high dosage, penicillin is non-toxic, and incidentally even *chronic* bone infection may be susceptible to penicillin in high dosage.

Penicillin-insensitive organisms—From the beginning it was known that the coli-typhoid, pyocyaneus and proteus bacilli were insensitive[1] to

[1] I have vivid recollections of a difficult bone-grafting operation performed during the recent war by one of my colleagues in which, at the end of a gruelling two hours, a bead of sweat dropped from his brow into the wound. After suitable expression of annoyance the wound was flooded with penicillin, only to become grossly infected with Ps. pyocyaneus which was flourishing in the penicillin solution.

One other case is significant. A moribund infant with diarrhœa, cachexia and pneumonia was given penicillin by infusion into the tibial medulla. The child survived; but osteomyelitis of the tibia with sequestration of nearly all the shaft was produced by the penicillin-infusion in which B. proteus and other penicillin-insensitive organisms were growing. This was an example of serious osteomyelitis produced by penicillin.

penicillin. What is more alarming is that many forms of staphylococcus which originally were destroyed by penicillin are now found to be insensitive. Barber and her co-worker[1] reported that before 1944 there were few penicillin-resistant staphylococci whereas, since then, no less than 10 to 20 per cent of all strains have proved to be penicillin-resistant. We have long known that the continued but sublethal application of antiseptics allowed only more resistant bacteria to survive so that succeeding generations acquired habituation. Similar resistance is becoming established to the antibiotics, and there is danger that unscientific treatment may produce new strains of insensitive bacteria. It is imperative, therefore, when infection of a wound is established, that bacteriological investigation should be completed at once in order that the sensitivity of the bacteria to various antibiotics may be determined. Appropriate chemotherapy should then be given in maximal dosage. It is futile to continue penicillin treatment if the organisms are penicillin-resistant, and it is dangerous to use any chemotherapeutic agent in sublethal dosage.

Streptomycin was developed from a strain of *actinomyces griseus* by Selman Waksman,[2,3] who was born in a Ukrainian town near Kiev and now works in New Jersey. He said that of ten thousand cultures of organisms studied in his laboratory, one thousand were found to have antibacterial properties, one hundred produced specific antibacterial substances when grown in liquid media, ten were isolated, and only one proved to be of therapeutic importance, namely, streptomycin. Streptomycin is outstanding for its effect on tuberculosis, particularly in the treatment of tuberculous meningitis. It also destroys bacillus coli, proteus vulgaris, pseudomonas pyocyaneus, clostridium Welchii and actinomyces. It should be given six-hourly by intramuscular injection with a daily dose of from one to two million units. Penicillin and streptomycin can be combined in the same injection. Streptomycin, like penicillin, is excreted by the kidneys and is valuable in the treatment of urinary infections. It passes through the placenta to the fœtus but does not diffuse easily into the cerebrospinal fluid. Intraspinal injections have therefore been used for cerebrospinal injections, but in a few cases such injection has caused paralysis of the cauda equina. Even after intramuscular injection neurotoxic effects involving the eighth nerve with transient deafness, vestibular disturbance and vertigo have been observed.

Habituation to streptomycin is established so readily that bacillus coli, vulgaris and pyocyaneus may become insensitive within forty-eight hours. Moreover, when resistance is established bacterial infection does not respond to a second series of injections even after the lapse of several weeks.[4] Streptomycin should therefore be given in maximal dosage, and in the treatment of pyogenic infection it should be discontinued after three or four days. If by that time it has not destroyed the bacteria that are sensitive it may well have created insensitivity by which they cannot be destroyed. On the other hand, the treatment of tuberculosis calls for long-continued dosage with streptomycin combined with para-amino-salicylic acid.

[1] Barber, M., and Rozwadowska-Dowzenko, M. "Infection by Penicillin-resistant Staphylococci." *Lancet*, 1948, 2, 641.
[2] Schatz, Albert, Bugie, Elizabeth, and Waksman, S. A. "Streptomycin, a Substance exhibiting Antibiotic Activity against Gram-positive and Gram-negative Bacteria." *Proc. Soc. Exper. Biol. and Med.*, 1944, 55, 66.
[3] Waksman, S. A., Bugie, Elizabeth, and Schatz. "Isolation of Antibiotic Substances from Soil Micro-organisms with reference to Streptothricin and Streptomycin." *Proc. Staff Meet. Mayo Clin.*, 1944, 19, 537.
[4] Knop, C. Q. "Experimental Study of the Development of Resistance to Streptomycin." *Proc. Staff Med. Mayo Clin.*, 1946, 21, 273.

Neomycin was described by Selman Waksman in 1950.[1] It promises to control the growth of the tubercle bacillus with less rapid habituation than is the case with streptomycin

Aureomycin, a yellow crystalline substance elaborated from *streptomyces aureofaciens*, was first described in 1948 by Duggar [2 3] It is of practical importance because it can be given by mouth and is absorbed readily from the alimentary tract, passing freely into body fluids and tissues, the cerebro-spinal fluid, and through the placenta to the fœtus Many organisms that are penicillin-resistant and streptomycin-resistant, or even streptomycin-dependent, are susceptible to its action The strains of staphylococci which since 1944 have become penicillin-insensitive are sensitive to aureomycin It must be recognised, however, that although the development of aureomycin-resistance is uncommon it has already been reported in a few cases of staphylococcal infection Aureomycin destroys all species of pathogenic cocci—staphylococcus, streptococcus, pneumococcus and meningococcus, and also bacillus coli and Freidlander's bacillus, but it has no proved action against clostridium Welchii or tetani, proteus vulgaris or pseudomonas pyocyaneus Considerable amounts remain unabsorbed and are excreted in the fæces The bacterial flora of the intestinal tract is changed completely and the fæces become odourless [4] Coliform organisms, streptococci and clostridia disappear , yeasts, micrococci and candida albicans remain alone, and there is in consequence some looseness of the stools Aureomycin may also cause nausea and vomiting. but in the usual dosage it has little toxicity The full dose for adults is 3 gm daily, given as two capsules each of 250 mgm four-hourly If there is nausea or vomiting one capsule should be given twice as often. The particular value of this antibiotic is in the control of penicillin-resistant staphylococci and the treatment of all forms of pneumonia.

Chloromycetin (chloramphenicol) was developed in the Parke-Davis Laboratories in Detroit from streptomyces isolated from soil in Venezuela—*Streptomyces venezuelæ* [5] It is the first antibiotic to be purified and produced synthetically on a commercial basis, the formula being of interest in so far as nitrobenzene and dichloracetic acid, which are recognised poisons, are included in the chain It is a neutral substance, equally active in acid and alkaline solutions, whereas penicillin which is acid, and even aureomycin which is basic, are most active only in acid solutions , and streptomycin which is basic, is active only in alkaline solutions Chloromycetin is given by mouth and is absorbed rapidly into the blood stream The list of pathogens which it inhibits is similar to that relating to aureomycin except that it is less active against the pyogenic cocci and more valuable in the treatment of infections by gram-negative bacilli, notably the enteric group, proteus vulgaris and pseudomonas pyocyaneus. After a loading dose of 3 gm , the same amount should be given daily for three days, divided into four doses each day, and thereafter half that amount daily for ten days

[1] Waksman, S A "Streptomycin and Neomycin" *Brit med J*, 1950, **2**, 595
[2] Duggar, B M "Aureomycin—a Product of the Continuing Search for New Antibiotics" *Ann N Y Acad Sci*, 1948 **51**, 177
[3] Bryer, M S, Schoenbach, E B, Chandler, C A, Bliss, E A, and Long, P H "Aureomycin, Experimental and Clinical Investigations" *J Amer med Ass* 1948, **138**, 117
[4] Dearing, W H, and Heilman, F R *Proc Staff Meet Mayo Clin*, 1950, **25**, 87
[5] Ehrlich, J, Gottlieb, D, Burkholder, P R, Anderson, L E, and Pridham, T G "Streptomyces Venezuelæ, the Source of Chloromycetin" *J Bact*, 1948, **56**, 467.

days when whole wards of patients succumbed from "hospital gangrene," through the period of the 1914-18 war when although elective surgery had become safe, a high proportion of soldiers developed gas-gangrene infection of their wounds, until the recent war when such infection was so rare that many of us never saw a single example. This remarkable progress has depended not so much on asepsis which aims at the exclusion of bacteria from the wound, or on antisepsis or antibiosis which aim at destruction of the bacteria, but on recognition of the importance of prompt excision of the wound with removal of all the devitalised tissues in which bacteria can grow. The frequency of gas-gangrene infection in the 1914-18 war arose from the fact that soldiers often lay in shell-holes for many days; whereas in the recent war with greater mobility and improved transport, men wounded on the European continent were operated upon in England within a few hours. Even to-day many wounds are contaminated with clostridia of the gas-gangrene group which, however, do not grow and multiply except in anaerobic conditions. This does not mean that the wound must necessarily be exposed to the external air, or that oxygen must be supplied by hydrogen peroxide irrigation; even freer oxygenation is available through the normal blood supply. If the walls and contents of a wound consist only of living pulsating tissue, the conditions are aerobic and gas-gangrene bacilli cannot survive. The fate of such bacteria is the clearest possible illustration of the important principle that soil is no less important than seed, and that early wound excision is no less important than chemotherapy.

Clinical signs of gas gangrene—The local signs are pain, swelling, crepitation of the tissues, and a curiously indolent, dry appearance of the wound but with a thin, watery foul-smelling exudate which escapes on gentle pressure. Only when the anaerobic infection is controlled does the discharge become frankly purulent, and herein lies the explanation of the "laudable pus" of an earlier generation of surgeons. The smell is characteristic; once recognised it is never forgotten. Three degrees of infection may be distinguished: (1) local infection of cellular tissues in the neighbourhood of the wound; (2) infection of one muscle or a group of muscles; (3) massive gangrene of all muscles and cellular tissues of the limb.

Surgical treatment—Immediate operative treatment is essential. The wound must be drained so freely, and relieved of tension so completely, that every tissue will regain a normal blood supply. All sutures must be removed and the wound should be extended in the long axis of the limb until it may even be ten or twelve inches in length. Every muscle that is gangrenous should be removed in its entirety, great care being taken to avoid damage to the blood supply of adjacent muscles. All wounds must be left widely open with gauze lightly packed between the walls. If there is massive gas gangrene immediate amputation is essential, the flaps being left open until the anaerobic infection is controlled. If the patient survives, secondary suture is usually safe after about ten days.

Antitoxic and antibiotic therapy—Until recent years the only available treatment apart from surgical intervention was antitoxin therapy. A polyvalent antitoxin including 50,000 units of the antitoxins of clostridia Welchii septicum and œdematiens was given intravenously and repeated as necessary. Radiotherapy was also advocated. It was then found that sulphonamides inhibited the growth of clostridium Welchii and septicum

but not œdematiens Much more powerful and specific chemotherapeutic agents are now available in penicillin, streptomycin and especially the newly discovered terramycin The clostridia are not susceptible to aureomycin or chloromycetin

Tetanus Infection

Anaerobic spore-forming clostridium tetani develops under conditions that are similar to those governing the growth of gas-gangrene clostridia[1] Three degrees of infection may be distinguished (1) local rigidity and twitching in the region of the wound, (2) generalised tonic rigidity, (3) recurrent generalised reflex spasms When infection is fully developed, trismus, dysphagia, risus sardonicus, opisthotonus and abdominal rigidity present a clear clinical picture If reflex spasms develop within twenty-four hours the prognosis is grave; if within two or three days the prognosis is fair, and if not until after the fifth day the outlook is good

Treatment of tetanus—Prophylactic protection against tetanus can be achieved by the active immunisation of tetanus-toxoid injections Moreover, as with gas gangrene, the development of tetanus can be prevented, or greatly modified, by early excision of the wound. If, nevertheless, infection becomes established 200,000 international units of tetanus antitoxin should be injected intravenously, with a further 50,000 units every seven days until the spasms subside. Streptomycin should be given by intramuscular injection and terramycin by mouth

Infection with Actinomyces

More than 80 per cent of actinomycotic infections in humans occur in the gastro-intestinal or respiratory tracts, but bone involvement has been recorded in the spine where it is often a direct extension from a focus in the lung or the paravertebral glands[2-4] Primary infection of wounds of the fingers after a human bite have been described by McWilliams[5] and Robinson[6] More recently Cullen[7] reported five cases of actinomycosis of gunshot wounds in a series of fifty compound fractures of the femur There was profuse and persistent discharge without radiographic evidence of obvious sequestra or foreign bodies, with frequent flares of cellulitis and abscess formation around the wounds, marked destruction of bone, general toxæmia and anæmia Discharge from the wounds persisted for more than twelve months Actinomyces were detected in fresh films of the pus and they were cultured Only in one case was the usual anaerobic actinomyces bovis isolated, in the other four the organism was aerobic, which is of interest because it has been believed that only anaerobic forms are pathogenic Treatment with potassium iodide gave little or no response, and only one case improved after radiotherapy At the time of Cullen's report the only antibiotic available was penicillin in small dosage and the response was disappointing It is now known that actinomyces are susceptible to both penicillin and streptomycin provided that they are given in full dosage

[1] Bonney, V, Box, C, and McLennan, J "Tetanus Bacilli recovered from Scar Ten Years after Post-operative Tetanus" *Brit med. J*, July 2, 1938 10
[2] Cope, Z "Actinomycosis" London Oxford University Press, 1938
[3] Tabb, J L, and Tucker, J T "Actinomycosis of the Spine" *Amer J Roentgen*, 1933, **29**, 628
[4] Dixon, G Joly "Generalised Actinomycosis with Predominant Spinal Symptoms" *Brit med J*, 1939, 2, 686
[5] McWilliams, C A "Actinomycosis of Phalanx of Finger" *Ann Surg*, 1917, **66**, 117
[6] Robinson, R A *J Amer med Ass*, 1944, **124**, 1049
[7] Cullen, C H "Infection of Gunshot Wounds with Actinomyces" Report to Brit Orth Ass, 1948

AFTER-TREATMENT OF WOUNDS

During the last ten years the after-treatment of wounds has developed in three well-defined stages (1) antiseptic treatment of the open wound, (2) closed plaster treatment of the open wound, (3) early closure of the wound by delayed primary suture or free-skin grafting

1) **Antiseptic treatment of wounds**—Formerly wounds were left open and dressed frequently, at least once or twice daily and sometimes more often Attempts were made to control infection and accelerate healing by a ritual of antiseptic therapy with coal-tar derivatives dyes, halogens, mercurial preparations, acridine compounds, cod-liver oil and many other



Fig. 413

Antiseptic treatment of infected wounds

The antiseptic treatment of wounds by dressings and irrigations (as, for example, by the Bunyan bag technique shown in this illustration) was replaced by the closed plaster method, and now by early closure of the wound by suture or grafting If skin has been destroyed it should be replaced at the time of the original operation of wound excision or, at the latest, within a week or ten days when infection has been controlled by chemotherapy

preparations [1,2] Granulating areas were cleansed with hydrogen peroxide, packed with bismuth-iodoform paste, flooded with Carrel-Dakin irrigations and enclosed in irrigating bags (Fig 413) It was even recommended that when a wound was excised the tissues should be scrubbed with soap and water and a nail-brush—a terrible heritage of the antiseptic era when the object of treatment was the destruction of bacteria at all costs, even at the cost of destroying living tissues Day after day, and week after week, the attack was continued Drainage tubes were inserted, packs were introduced, taken out and replaced and antiseptic solutions were applied incessantly

It is true that staphylococci are susceptible to violet dyes, that hæmolytic streptococci are inhibited by acridine compounds, and that pyocyaneus is destroyed by weak acetic acid, but violet dyes, which arrest the growth of staphylococci, have no action on gram-negative bacilli; acridine compounds

[1] Garrod, L P "Action of Antiseptics on Wounds" *Lancet*, 1940, 1, 845
[2] Lichtenstein, M "Cod-liver Oil Dressings" *Lancet*, 1939, 2 1023 (With bibliography)

are useless in staphylococcal infections, and acetic acid controls only the growth of pseudomonas pyocyaneus, which flourishes in nearly all other antiseptics These selective actions were seldom recognised, and antiseptics were used indiscriminately with little regard to the bacteriological findings Moreover, they could be applied only to the surface of a wound, and even Carrel-Dakin tubes placed in the recesses of a wound cavity were still on its surface, whereas bacteria were multiplying in deeper layers, in fascial planes and in intermuscular spaces. Even the control of surface infections was lost within about forty-eight hours because the continuous application of any noxious agent that is not wholly lethal allows more resistant bacteria to survive, and succeeding generations lose all susceptibility Thus the treatment of wounds by frequent dressing and antiseptic applications was characterised by the slow healing of granulation, complicated almost invariably by secondary infection

2) **Closed plaster treatment**—The danger of secondary infection was proved by Winnett Orr,[1] who demonstrated that when the tension of inflammatory exudates within an infected region was relieved by free incision, healing was more rapid if the wound was enclosed in plaster than if it was left open for frequent dressing The danger was expressed graphically in the *Lancet*[2]. "Frail capillary endothelium tears readily and waits with pouting mouth for further organisms, a dressing with the gentlest hands is then like the trampling of an elephant's feet" The merit of enclosure of a drained wound in plaster depended not only on immobilisation, protection from further injury, and lymphatic stasis which minimised spread of infection[3 5] but, above all, on the fact that repeated dressing of the wound was impossible.

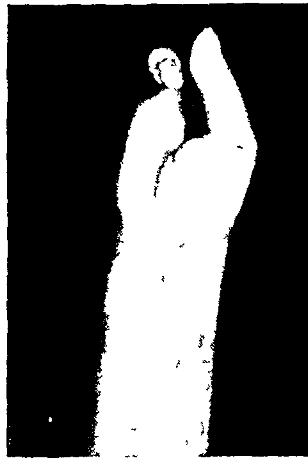
The same principle was applied by Trueta[6] to the emergency treatment of contaminated wounds After excision of dead and dying tissue, gauze was laid between the walls of the wound and the limb was immobilised in plaster If there was no infection healing took place within a few weeks; and even when there was infection, healing was not complicated by secondary contamination This treatment was used after excision of contaminated wounds and drainage of infected wounds, after sequestrectomy and drainage of bone abscesses, in the treatment of infected amputation stumps, and in the management of burns (Figs 414-428)[7] The technique placed emphasis on the importance of excising dead tissue, draining infected areas and immobilising injured tissues, and it proved the futility of repeated dressing with antiseptic applications, but healing was still by granulation and it was delayed, the scars were broad, adherent and often unstable.

3) **Early closure by skin suture and grafting**—The third stage in the development of wound treatment is based on the same principles of excision of contaminated wounds, drainage of infected wounds, immobilisation of injured tissues and prevention of secondary infection, but whereas local antiseptic treatment of infection failed in former years, antibiotic control has now succeeded and wounds can be closed quickly and completely by delayed primary suture of the skin, or by the application of free-skin grafts, so that the penalty of healing by granulation is avoided

[1] Orr, H Winnett "Osteomyelitis and Compound Fractures" London Henry Kimpton, 1929
[2] "Rest for Inflamed Tissues" (Leading Article) *Lancet*, 1941, 2, 641
[3] Trueta, J, Barnes, J M "Immobilisation in Treatment of Infected Wounds" *Brit med J*, 1940, 2, 46
[4] Trueta, J "Treatment of War Wounds and Fractures" London Hamish Hamilton Medical Books, 1939
[5] Trueta, J, and Barnes J M "Absorption of Bacteria and Toxins from the Tissues" *Lancet*, 1941, 1, 623
[6] Trueta J "Closed Treatment of War Fractures" *Lancet*, 1939, 1, 1452, 1939 2, 1173
[7] Cohen, S M, and Schulenberg, C "War Wounds of the Limbs" *Lancet*, 1940, 2, 351


Fig 414


Fig 415


Fig 416

Closed plaster treatment of contaminated wounds

Explosive wound of the hand, three fingers were blown off and the fifth finger was shattered After wound excision the closed plaster treatment was used The fractures united, the wound healed by granulation and useful function was regained (*Mr S Cohen's case*)


Fig 417


Fig 418


Fig 419


Fig 420

Closed plaster treatment of infected wounds

A gangrenous limb (Fig 417) was treated by circular amputation and the application of a closed plaster, so that it was possible to save a below-elbow stump despite the extensive destruction of skin (*Mr S Cohen's case*)



FIG 421
At the time of operation



FIG. 422

Closed plaster treatment of infected joints (Figs. 421-424)

Penetrating wound of the knee joint complicated by septic arthritis and treated by free incision into the front of the joint and suprapatellar pouch, the postero-medial and the postero-lateral compartments (Fig 421) The wounds were lightly packed open with vaseline gauze and a plaster spica was applied (Fig 422) After five weeks, when the plaster was



Fig 423
Five weeks after operation



Fig. 424
Eleven weeks after operation.

changed, the wound looked unpleasant (Fig 423), but the patient's general condition, and the temperature and pulse chart, showed that the infection was controlled Six weeks later the wound was almost healed (Fig 424) Immobilisation in a plaster spica was then continued until the joint was soundly ankylosed



FIG. 425

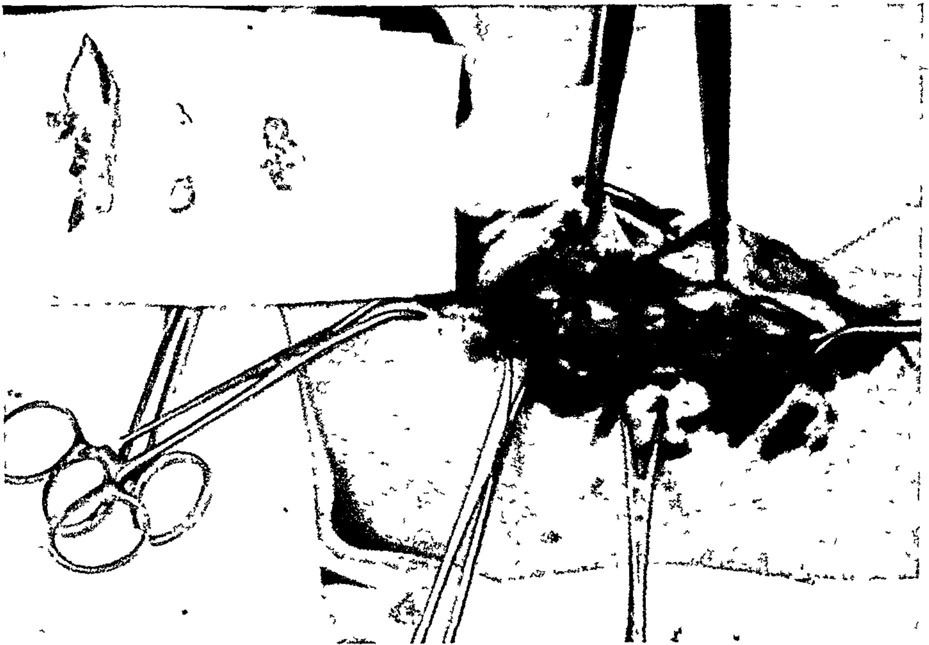


FIG 426

Closed plaster technique after sequestrectomy (Figs. 425-428)

Compound fracture of tibia with sequestration of a large part of the shaft (Fig 425) The operation is performed by soft-tissue dissection alone, there is no scraping, and if possible no chiselling or gouging The walls of the wound are kept



Fig 427



Fig 428

lightly apart by loose "packing" with vaseline gauze Plaster is applied The plaster is heavily stained and there is often a foul smell (Fig 427) Nevertheless the wound is healing beneath the plaster In this case, six weeks after operation, at the first change of plaster, the wound was almost completely healed (Fig 428) Plaster immobilisation was continued until the fracture was united

Fig. 429



Fig 430

Technique of split-skin grafting

A split-skin graft from the antero-medial aspect of the thigh is spread on tulle-gras Surface granulations are shaved off the recipient area and, after hæmorrhage has been stopped by adrenaline packs firmly pressed on the wound, penicillin-sulphathiazole powder is gently massaged in The graft is then applied with the firm pressure of a crêpe bandage over many layers of wool

(5) RESTORATION OF SKIN COVER BY DELAYED SUTURE OR FREE-SKIN GRAFTING

Delayed primary suture—The more quickly skin cover is gained the more certainly is secondary infection avoided If the wound has been excised within a few hours of injury, and local and general signs show no evidence of infection, it should be sutured on the third or fourth day. It is unwise to wait longer because the elasticity of skin encourages quick retraction, and late secondary suture necessitates under-cutting of the skin margins and suture under tension After delayed suture, chemotherapy with penicillin or one of the other antibiotics should be continued for another ten days

Replacement of destroyed skin by free grafting—Delayed primary suture is indicated only when the surface area of a wound arises solely from



FIG 431

An extensive granulating wound involving the whole circumference of the leg has been skin-grafted This shows the stage of healing three weeks after operation The few areas where grafts have not survived can be re-grafted at a second operation

retraction of the skin margins (see Fig 399), it is not indicated when there has been actual destruction If a considerable area of skin has been destroyed it is unwise to drag the margins of a wound together under tension, even if distant "tension incisions" are made, with approximation at the cost of gaping at these secondary incisions Similarly, rotation flaps and advancement flaps, which aim at covering the wound with whole-thickness skin while leaving other areas to heal by granulation, are unsafe at this early stage when there is still traumatic œdema and the possibility of latent infection Sloughing of skin that has been sutured under tension, or of flaps that have been advanced, is so likely to occur that the difficulties may be increased It is wiser to replace destroyed skin by free split-skin grafts which may be applied safely even at the time of the original operation of excision or certainly within seven to ten days (Figs 432-435) A large part of the transplanted skin will live—perhaps all of it—and even if some areas fail

to survive nothing has been lost; free grafting can be repeated a second or even a third time The purpose is to give skin cover as quickly as possible. The best surgical dressing for a wound is skin.

Split-skin grafting—A free graft of skin (Ollier-Thiersch graft, razor graft, intermediate skin graft or split-skin graft) is cut as a sheet consisting of at least half the thickness of the skin, using a Blair knife, Humby knife or Padgett dermatome The graft does, of course, depend for survival on nourishment from the recipient area, and it cannot live unless there is close contact with a vascular bed of granulation tissue or muscle It must be maintained in firm apposition with its bed by the pressure of wool dressings



FIG 432

Grenade explosion in the hand destroying a large area of skin, together with the extensor tendons and the metacarpo-phalangeal joint of the index finger. The wound was excised and immobilised in plaster for ten days, throughout which time the limb was elevated (See Figs 433-435)

and crêpe bandage, if it is lifted by serous exudate, pus or blood it will not survive Moreover, a free graft cannot live on a bed of bare bone, tendon or avascular scar tissue Nevertheless when bone is exposed, and even when there is a discharging sinus, granulating areas should be grafted up to the margins of the bone or the sinus.

Regeneration of the area from which the graft is cut takes place from epithelial cells in the hair follicles and glands of the dermis, and it is usually complete within about ten days A simple dressing with tulle-gras is all that is needed A scar remains, particularly in the recipient site, but this can be treated later by excision and suture, or by transferring a pedicled flap of whole-thickness skin.

Pinch grafts—The ugliness of both donor and recipient scars is even greater when pinch grafts are used. A pinch graft consists of a small island



FIG 433

Same case as Figure 432 On the tenth day the granulating area was skin-grafted with patch grafts Figure 433 shows the condition one week after grafting



FIG 434



FIG 435

End result in the case shown in Figures 432-433 The index metacarpo-phalangeal joint was stiff and there was slight contracture in the web of the thumb, but otherwise function was perfect (*Treated by Wing-Commander A Butler, now orthopaedic surgeon in Montreal*)



FIG 436

Open fracture of the shaft of the tibia twelve months after injury Union has been delayed by the failure to remove sequestra which caused sinus formation and persistent low-grade infection



FIG 437



FIG 438

Same case as Figure 436 After sequestrectomy the sinus healed at once, the fracture would then have united by continued immobility alone—but it would have been *slow* (Fig 437) Bone grafting was therefore performed after three months (Fig 438)

of skin, full thickness at its centre and thin at the periphery, which is cut by lifting a cone of skin with the point of a straight needle and cutting through the base with a scalpel The pinches of skin are placed on the donor site at intervals of one centimetre The technique is useful when wounds are so extensive that even the skin of the whole abdominal wall and both thighs is not sufficient as a donor area It is also valuable in that pinch grafts can be taken under local anæsthesia without moving the patient from his bed. Even in these circumstances the grafts should be taken only from areas that will not be needed later as donor sites for other types of graft

Instability of scars resulting from split-skin grafting—The scars resulting from split-skin grafting and pinch grafting are usually thin and unstable, and they may not be strong enough to heal without sloughing if incisions are made through them for later reconstructive surgery Nevertheless, by such grafting the first purpose is achieved—namely, immediate skin cover with control of infection The next object can be achieved by later replacement of the unstable scar with whole-thickness pedicled flaps (page 277)

6 FINAL CONTROL OF INFECTION BY SEQUESTRECTOMY

Even when every attempt has been made to prevent infection of the wound by prompt excision, continuous chemotherapy, and early restoration of skin cover, a sinus may remain One or more foreign bodies may have been overlooked at the first emergency operation, or bone which at that time appeared to be alive may have necrosed and separated as a sequestrum Radiographic examination reveals opaque foreign bodies and fragments of sequestrated bone which are distinguished from adjacent living bone by their relative density (Fig 436) The sinus must be explored and the foreign body or sequestrum removed, because so long as discharge continues there will be low-grade infection with delay in union of the fracture Moreover, secondary reconstruction or bone-grafting operations cannot be undertaken safely until infection is quiescent and the wound is soundly healed

The importance of early sequestrectomy—*and the exception to the rule*—Sequestrectomy is often followed by striking acceleration in the recalcification of callus, and in the rate of union of a fracture which had been indolent for months It is important, therefore, to remove dead bone as soon as it is separated from living bone and certainly within two or three months of injury There is, however, one exception to this rule If the sequestrum consists of the whole thickness of the shaft of a long bone, its removal should be deferred until sufficient bone has been laid down as an involucrum to prevent collapse of the periosteal tube It is the same principle of treatment that applies to the total excision of all bone fragments at the time of wound excision, and to the treatment of acute osteomyelitis by diaphysectomy. Any operation that allows collapse of the periosteal tube and obliteration of the subperiosteal hæmatoma is likely to cause non-union, with a wide gap between the fragments, often necessitating a difficult bone-grafting operation and sometimes causing serious shortening of the limb It is not, however, necessary to wait for the involucrum to be complete and for the bone to unite round the sequestrum, this might well involve a delay of twelve months or two years As soon as subperiosteal ossification is evident in the radiographs the operation can be performed.

Fig. 439

Fig 440

Fig. 441

Fig. 442

A pilot baled out too late and fractured his femur. Four inches of the proximal fragment was driven into ploughed field, it lost its blood supply and sequestrated (Fig. 439). It was not removed until a firm involucrum had formed (Fig. 440) By immobilisation alone the fracture then united soundly.



Fig 443

Despite immobilisation for twelve months the patient regained more than 90° of knee movement and went back to duty—flying many hundred hours in R A F Coastal Command

Even when four or five inches of the shaft of the femur must be removed, sound union of the fracture can be secured without shortening provided that special care is taken to immobilise the region continuously and without interruption (Figs 439-445) It is particularly important to maintain this continued protection throughout the times of operation and of every subsequent redressing or replaster A single careless movement breaks the soft callus and, if movement is repeated whenever the plaster is changed, the fracture will never unite Before operation the patient is fixed in position on a traction table, with a strong pull from a skeletal pin in the bone to the traction screws of the table The site of fracture is supported



Fig 444



Fig 445

Same case as Figures 439-443 Figure 444 shows the wound at the first change of plaster one month after sequestrectomy Figure 445 is at the second change of plaster only one month later Large wounds due to retraction of skin (without destruction of skin) heal rapidly under plaster as soon as infection is controlled

to prevent even momentary backward sagging While traction and fixation are maintained, the sequestrum is removed, the wound is packed lightly with vaseline gauze, and a double plaster spica is applied incorporating the traction pin Two or three months later when the plaster is to be changed, the skeletal pin is again fixed to the traction table and the thigh is supported while the plaster is changed It may be necessary to continue the protection uninterruptedly for six months or longer but, if care is taken to avoid any single movement, the fracture will unite

Unusual foreign bodies causing sinuses—Sometimes a sinus continues to discharge even when there is no radiographic evidence of a foreign body or sequestrum It should still be explored because the foreign body may consist of wood, leather or clothing which cast no radiographic shadow (Fig 446). A museum collection of foreign bodies removed from wounds would rival the surgical collections of our forefathers—it would include fragments of brick, bits of glass, pieces of telegraph pole, sections of roof-laths and plaster, tunic buttons, fragments of leather belts, and screws, nuts, bolts or other parts of the fuselage of aeroplanes In the first great war, three metallic foreign bodies removed from the buttock of a patient proved to be golden sovereigns. After a London blitz in the recent war



Fig 446

Large fragments of wood, one measuring four inches in length, removed from a wound which continued to discharge through sinuses although there was no radiographic evidence of foreign bodies or sequestra

a fragment of crockery removed from the depths of a sinus was marked clearly "— de Paris", it was a relic of the demolished *Café de Paris* From another wound of the thigh a large piece of sequestrated bone was extracted, but there was no fracture of the femur or other evidence of a source from which the bone could have derived, it was in fact part of the skeleton of another victim of the same air-raid

Sequestrectomy is not a "scraping operation"—When some of us were young we saw long lists of operations displayed on hospital notice-boards: (1) scraping of sinus, (2) scraping of sinus, (3) scraping of sinus—and so on for a list of ten or twelve scrapings of sinuses The frequency of the operation arose from the fact that such scraping produced more sinuses than it cured. A curette or spoon is not needed, a gouge or chisel should seldom be used Ideally, the only instruments required are a scalpel and a pair of forceps. If every sequestrum or foreign body can be exposed by soft-part dissection and lifted out without chiselling of bone the wound will heal by first intention.

7 REPLACEMENT OF UNSTABLE SCARS BY WHOLE-THICKNESS SKIN GRAFTS

The stages of treatment outlined so far have controlled infection, but it may be that destroyed skin has been replaced by free grafting so that the scar is unstable, and if it lies over an avascular bed, as, for example, the subcutaneous surface of the tibia, it is liable to ulcerate and cause minor infection with delay in consolidation of the fracture Only when adherent and ulcerating scars are replaced by freely mobile skin is safety assured Moreover, if the fracture is malunited or un-united, and reconstructive bone operations are needed, success will be imperilled by incisions made through thin scars which are likely to break down The scarred area must first be

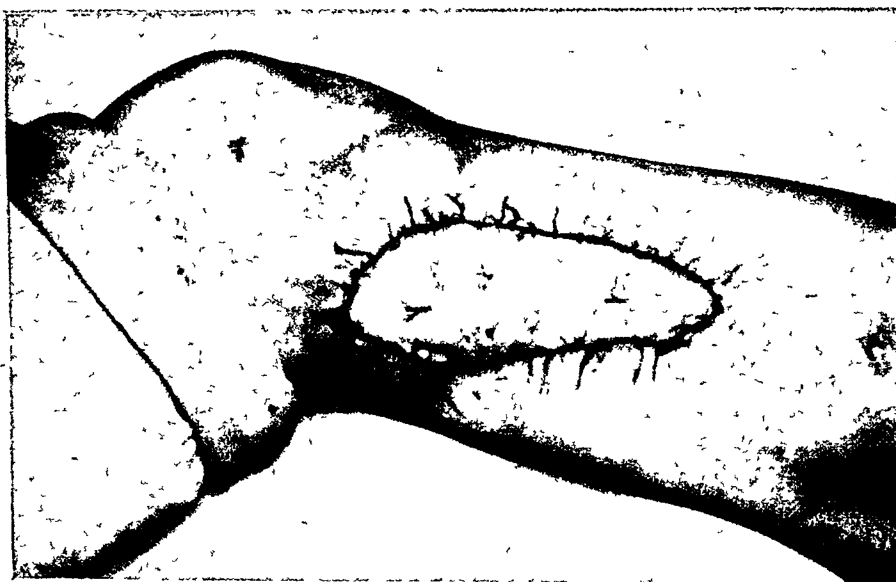


Fig 447

Whole-thickness skin may sometimes be transferred as a free graft, especially thin skin from the thigh dissected carefully to include no fat But even then there is danger of sloughing, and moreover a pedicled whole-thickness flap, including some fat, gives sounder cover with a better appearance and greater mobility of the skin

replaced by whole-thickness skin, freely mobile and not adherent, transferred either by pedicle tube flaps or by abdomen–arm or cross-leg flaps

Pedicle tube flaps—It is sometimes necessary to raise a tube of skin on the abdomen and after several weeks transfer one end to the patient's wrist, when the blood supply from the wrist is sufficient to maintain nutrition of the tube the other end is detached from the abdomen and attached to the recipient site, and finally, when a blood supply from the recipient site is assured the tube is opened to a flat surface of skin and stitched to the area from which the unsound scar has been excised This technique calls for the special skill and experience of plastic surgeons But other methods of direct transference of pedicled flaps from the abdomen to the upper limb, or from one lower limb to the other are within the capacity of every surgeon who treats fractures

Abdomen–arm flaps—In the upper limb it is easy to replace unsound scars by the simple two-stage operation of abdomen–arm flaps The area



Fig. 448



Fig. 449

Repair of an unstable scar on the foot (Fig 448) by cross-leg flap After the flap has been designed and marked out on the donor limb it is raised with a wide base and sutured to the raw area left after excision of the unstable scar (Fig 450) The limbs are held in contact by means of plaster and after three or four weeks the base of the flap is divided. The donor area is made good by secondary suture or by free split-skin grafting.

to be replaced is first mapped and suitably marked on such part of the skin of the abdominal wall as will permit a comfortable position during the three or four weeks when the limb must be held in close contact with the abdomen The blood supply of the flap is preserved best if its width is greater than its depth, so that it is elliptical or lunate in shape rather than semicircular or tongue-shaped The base of the flap should usually measure twice its depth and certainly the proportion should be not less than three to two The scar on the upper limb is excised and the margins are undercut sufficiently to permit easy suture of the donor flap. The flap is then raised from the abdomen, left attached by its broad base, and sutured to the area from which the scar has been excised (Fig. 465). The abdominal wound is covered with



Fig 450

End of the first-stage operation of cross-leg skin transference (Figs 448-449) Three weeks later the base of the flap is divided and the donor area is made good by suture or split-skin grafting

vaseline gauze and the limb is held securely to the trunk by many crêpe bandages, or even a plaster cast, so that the pedicled base of the flap will not be twisted or strained After three or four weeks, by which time the flap has gained a free supply of blood from the limb, its base is divided and repair of the scarred area is completed by accurate suture The granulating surface on the abdomen is closed by undercutting and approximating the skin margins.

Cross-leg flaps—The principle of transferring flaps of skin from one lower limb to the other is basically the same as in the transference of abdomen–arm flaps Ideally the base of the flap should measure twice its depth and the area of attachment to the recipient limb should be not less than 60 per cent of its total area The scar to be replaced is measured accurately by cutting a pattern from an overlaid piece of jaconet or tin foil, allowance being made for the increase in size of the lesion that always occurs

when scar tissue is excised. Additional allowance is then made for the bridge that will span the distance from donor limb to recipient site—in other words, the flap to be raised must equal the width of the deficiency and the depth of the deficiency together with the length of bridge. The pattern is then outlined on the donor limb in a site that will permit a comfortable cross-leg position for not less than three weeks, care being taken to avoid encroachment on the region of the tendo Achillis or the subcutaneous surface of the tibia. Flaps from the opposite calf will cover lesions of the subcutaneous surface of the tibia (Figs 455-456) or the lateral aspect of the leg and foot ; and flaps from the lateral aspect of the donor limb will cover lesions of the medial side of the foot, ankle or lower leg.[1]

When the scar has been excised and the flap is sutured in position the raw area left on the donor limb may be covered with a free split-skin graft from the thigh, or be simply dressed with vaseline gauze with the object of closing it after three weeks by secondary suture. The limbs are held in their cross-legged position by means of a plaster cast. After three or four weeks the base of the flap is divided ; repair of the recipient site is completed by accurate suture ; and the donor site is made good by secondary suture or split-skin grafting.

Delayed transference of flaps—Sometimes the lesion to be repaired is of such shape and size that it is impossible to raise a donor flap that is twice as broad as it is deep. In these circumstances the principle of delayed transference should be used. Even a tongue-shaped flap can be transposed if at a preliminary operation it is only partly raised, with an intact bridge of tissue at its apex as well as at its base. After fourteen days the apex is divided with some assurance that hypertrophy of vessels through the base will so ensure its vitality that it can be transferred to the new site with safety. There is little merit in complete elevation of a flap with resuture to its original bed : if it is safe to raise a flap completely it is safe to transfer it at once.

8 LATE RECONSTRUCTIVE SURGERY

In this chapter the management of open fractures has been discussed in sections corresponding in their sequence to the timing of open-fracture treatment, namely : first-aid and resuscitation ; excision of the wound ; chemotherapeutic control of infection ; early restoration of skin-cover by delayed suture or free grafting ; final control of infection by sequestrectomy ; replacement of unstable scars by pedicled flaps , and then—*and only then*—reconstructive surgery with plates, screws and whole-thickness bone grafts. Many disasters have occurred because surgeons have tried to jump the fourth fence before clearing the second , because they have used plates and screws before infection was controlled ; and because fractures have been exposed for late reconstruction through skin that was so thin and unstable that it broke down and caused recurrent infection, whereas it should first have been replaced by whole-thickness pedicled flaps. A few case-reports have been selected to illustrate the sequence of the various stages of treatment. Some are personal cases treated in civilian practice and in the orthopædic service of the Royal Air Force ; but many others were dealt with by surgical colleagues and friends who were responsible for the treatment of soldiers and sailors as well as airmen.

[1] Brathwaite, F., and Moore, F. T. "Skin Grafting by Cross-leg Flaps." *J. Bone Joint Surg.*, 1949, 31-B, [illegible]

CASE 1—ILLUSTRATING ALL STAGES OF TREATMENT OF A SERIOUS OPEN FRACTURE—RESUSCITATION, TRANSFUSION, FREE-SKIN GRAFTING, SEQUESTRECTOMY, CROSS-LEG FLAP, DOUBLE ONLAY BONE GRAFT (FIGS. 451-462)



Fig 451



Fig 452

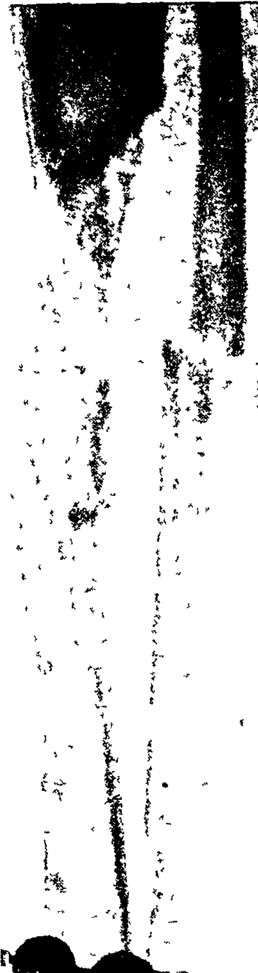


Fig 453

Case 1—A shell wound destroyed more than half the skin of the leg and shattered the tibia There had been serious hæmorrhage, and blood poured from the granulations every time the wound was dressed (Figs 451-452) Seven pints of plasma and blood were transfused at the primary treatment and in later stages The limb was supported on a Braun's splint with skeletal traction from the lower tibia The extent of sequestration of the tibia is seen in Figure 453, more than half the length of the shaft of the bone was destroyed



FIG. 454

Case 1 (*continued*)—Split-skin grafts were applied to the granulating surfaces After four weeks the sequestra shown in Figure 453 were removed Primary healing of the granulating wound was then completed by a second free-skin grafting operation



FIG 455



FIG. 456

Case 1—Chemotherapy with penicillin had been continued and after completion of the second free-skin grafting operation infection was controlled. The unstable scar was then replaced by a two-stage cross-leg flap transference (Fig 455) Two months later it was safe to expose the tibial fragments by an incision through the transferred flap (Fig. 456).



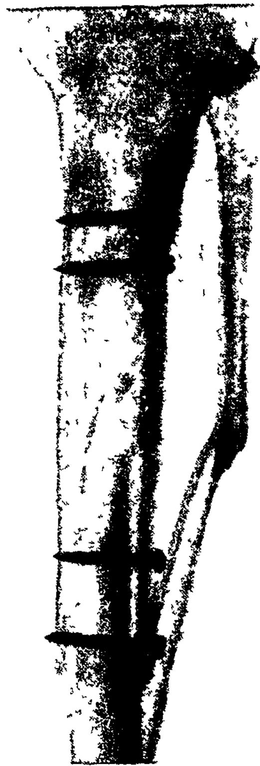




Fig 458

Fig 457

Fig 459

Case 1 (*continued*)—Double onlay grafts from the opposite tibia were fixed by means of vitallium screws, and the gap between the tibial fragments was packed with cancellous bone chips cut from the ilium (Fig 457) There had also been a fracture-dislocation of the talus of the opposite ankle (Fig 458) which was reduced by manipulation (Fig 459)



Fig 460



Fig. 461

Case 1—The patient was a Canadian and he not only played baseball as skilfully as before but went back to full operational duty in an A1 category within twelve months of his injury. (*Treated by Mr Lawson Dick, now of Edinburgh, when in the Orthopædic Service of the Royal Air Force*)

CASE 2—SEVERE WOUND OF THE FOREARM TREATED BY ABDOMEN-ARM SKIN TRANSFERENCE (FIGS. 462-466)



FIG 462



FIG 463



FIG 464



Fig 465



Fig. 466

Case 2—A cannon shell blew out the upper third of the ulna together with a wide area of skin and muscle (Figs 462-464) The wound was excised and immobilised in a closed plaster Three weeks later a flap of whole-thickness skin was transferred from the loin (Fig 465) The final result was amazingly good The patient regained a normal range of movement at what amounted to an arthroplasty of the joint There was no power of extension against resistance but flexion movement was very powerful and there was little instability (*Treated by Wing-Commander Morley at Royal Air Force General Hospital, Ely*)



Fig 469

Case 3—A mortar-bomb wound destroyed much of the skin of the forearm and shattered the lower shaft of the radius There was severe hæmorrhage and an emergency tourniquet had been applied A plasma drip was started at once, and after admission to hospital two pints of blood were transfused The wound was excised and the severed radial artery was ligated The limb was first supported by means of a plaster slab and then immobilised in a complete plaster cast. Seven months later there were persistent sinuses from sequestration of bone (Fig 467) The sequestra were removed The unstable scar was then replaced by a two-stage abdomen–arm skin flap transference (Fig 468) Three months later the dislocated lower end of the ulna was excised and the defect in the radius was bridged by an onlay graft with four vitallium screws Low-grade infection recurred with the formation of a sinus and, for this reason, the screws were removed four months after grafting (Fig 469) Normal function was regained

CASE 4—OPEN FRACTURE OF RADIUS—INFECTION, SEQUESTRECTOMY, BONE GRAFTING (FIGS. 470-474)



Fig. 470





Fig. 471



Fig. 472



Fig. 473



Fig. 474

Case 4.—After an open fracture of the radius from motor-car injury there was persistent infection with some [illegible] of bone causing delayed union (Fig. 470). The sequestra were removed and the sinuses then healed (Fig. 471). Three months later the fracture was bone grafted with arthrodesis of the inferior radio-ulnar joint (Fig. 472). Normal movement was regained (Figs. 473, 474).

CASE 5—SHELL WOUND IN WHICH IT WAS IMPOSSIBLE TO REMOVE ALL METALLIC FRAGMENTS (FIGS. 475-478)



Fig 475





Fig 476

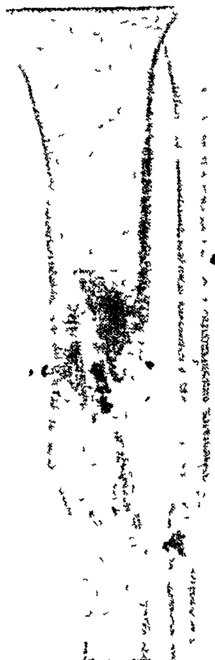


Fig 477



Fig 478

Case 5—Open fracture of the tibia from shell wound with diffuse spattering of tissues by metallic fragmen (Fig 475) The wound was excised but it was impossible to remove every foreign body (Fig 476) Neverthele the wound healed promptly and the fracture united by immobilisation alone, without necessity for bone graftin Figure 478 shows the patient learning to jump from a height—an important part of rehabilitation

CASE 6—SHELL WOUND DESTROYING LARGE AREA OF SKIN AND PART OF THE SHAFT OF THE RADIUS ABDOMEN-ARM SKIN TRANSFERENCE. BONE GRAFTING OF RADIUS WITH INFERIOR RADIO-ULNAR ARTHROPLASTY (FIGS. 479-484)



FIG. 479



FIG. 481



FIG. 480

Case 6

A shell wound destroyed a large area of skin of the forearm and there was a comminuted fracture of the shaft of the radius The wound had been excised, and it healed by granulation with minimal infection, but the radius was un-united, the lower end of the ulna was dislocated (Fig. 480) and the scar was thin and unstable The scarred area was excised and replaced by whole-thickness skin transferred from the abdomen in a two-stage operation. Figure 479 shows the limb after completion of this skin transfer.



Fig 482



Fig 483



Fig. 484

Case 6 (*continued*)

The first step in the operation of surgical reconstruction was excision of the lower end of the ulna so as to permit of realignment of the shortened radius (Fig 481). The gap fracture of the radius was then exposed and the fragments were freshened An onlay graft cut from the tibia was fixed with four vitallium screws (Fig 482) The gap was filled with iliac cancellous chips (Fig 483) The fracture united soundly in good position with normal movement of the wrist and radio ulnar joints (Fig 484)

CASE 7—MULTIPLE WOUNDS AND OPEN FRACTURES OF ALL FOUR LIMBS (FIGS. 485-487)



Fig 485



Fig 486



Fig. 487

Case 7—An air-gunner sustained contaminated wounds of all four limbs with open fractures of the metatarsals of the left foot, hallux of the right foot, left tibia and fibula, and both femora (Figs 485-486) In primary resuscitation he was given two pints of plasma The wounds were excised The feet and legs were immobilised in plaster, and the femora were treated in Thomas' splints by skeletal traction from the tibial tuberosities After eighteen weeks in hospital he enjoyed himself in a rehabilitation centre He then had movement of the left knee from 180° to 70°, and of the right knee from 180° to 60°, but perhaps he had not done very well because he lost this race—he is the man on the right He returned to full duty in seven months.

CASE 8—MULTIPLE INJURIES—FROSTBITE, OPEN FRACTURES, DISLOCATIONS (FIG. 488)

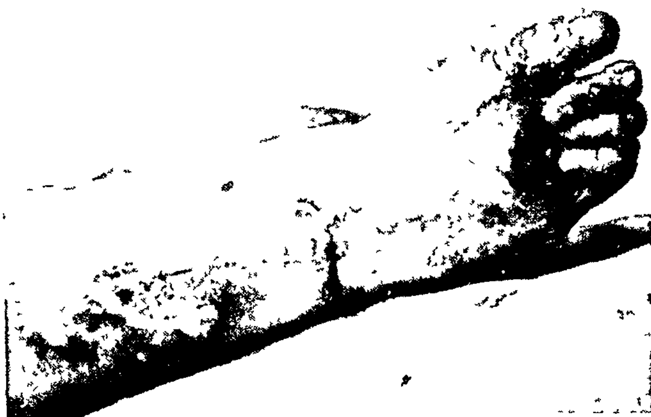


FIG 488

Case 8—A young pilot was flying in Scotland and the last thing he remembers is that visibility closed down He recovered consciousness twenty-four hours later The wreckage of his aircraft, which had been flown into the side of the mountain, was a hundred yards distant, he had been flung clear He had a dislocated shoulder and a broken neck When he looked down he saw the ends of his shin sticking out through his flying suit The wound was bleeding and he thought he would bleed to death, so he took off his neck-tie and tied it round the knee as a tourniquet This caused the worst of his injuries, because he lay on the mountain-side for three days and nights in midwinter before being discovered by two deer-stalkers By then he had gangrene of the toes and forefoot The wound of the leg was excised, fragments of heather were removed from its depths. the forefoot was amputated, the dislocated shoulder was reduced, and the fractured cervical spine was immobilised in plaster Within twelve months he was flying aircraft again

CASE 9—MULTIPLE INJURIES. PERHAPS THE RECORD! (FIG. 489)

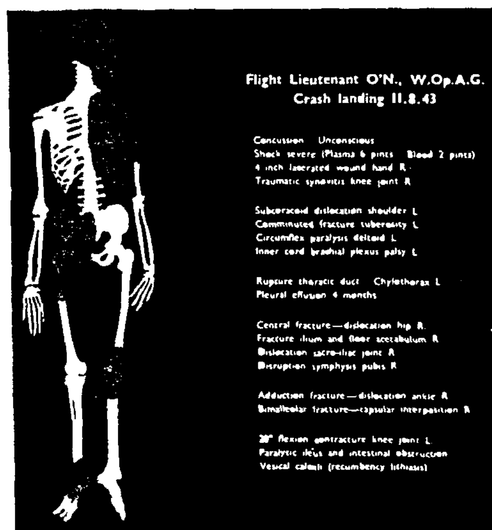


FIG 489

Case 9—In a crash landing Flight-Lieutenant O'N. sustained a number of major injuries There was dislocation of the left shoulder with fracture of the great tuberosity and circumflex palsy, and an incomplete traction lesion of the inner cord of the brachial plexus Rupture of the thoracic duct caused chylothorax with effusion for two months There was a central fracture-dislocation of the right hip joint with comminution of the floor of the acetabulum associated with disruption of the pelvis, dislocation of the sacro-iliac joint and separation of the symphysis pubis This was complicated by paralytic ileus There was an adduction type of fracture-dislocation of the right ankle, and rupture of the ligaments of the left knee causing flexion contracture which proved difficult to correct As convalescence was approaching he developed vesical calculi which had to be removed Is this a record for survival after multiple injuries? Almost certainly not—but nevertheless the case is surely a record The flight-lieutenant was back on flying duty in ten months, and he was fighting in France with a full operational category in eighteen months (Crash landing on August 11, 1943 He resumed non-operational flying on June 10 1944 He went back to operational flying on February 26, 1945)

AMPUTATIONS FOR OPEN AND INFECTED FRACTURES

Indications for immediate amputation—Amputation is seldom needed in the treatment of bone and joint injuries, and the decision to adopt this unusual and drastic measure should always be supported by a second opinion. *If the main blood vessels are not destroyed and there is no irrecoverable nerve lesion*, the limb should be saved no matter how severe the contamination of a wound, the comminution of a fracture, or the destruction of skin Even serious infection may be controlled by chemotherapeutic measures, skin destruction over the whole circumference of a limb can be treated successfully by skin grafting; and if immobilisation is continued and prolonged, contaminated wounds heal and shattered bones unite Almost the only indication for immediate amputation is destruction of the main blood vessels and the imminence of gangrene.

Technique of immediate amputation—Within about eight hours of injury, bacteria have not invaded deep tissues and lymphatics, and infection is not established. If the wound is so remote from the site of amputation that all dead and contaminated tissue will be removed, and if it is reasonably certain that the patient will remain under observation for several days after operation, a formal flap amputation may be performed at the site of election with immediate suture of the skin flaps. If more than about eight hours have elapsed and the amputation must be performed through wounded tissues, or if early evacuation of the patient may become necessary, delayed primary or secondary skin suture should be employed as in the technique of late amputation.

The site of election and the minimal length of stumps are illustrated in Figures 490-493 The ideal stump is firm conical in shape so that it will fit the bucket of an artificial limb, and free from skin folds which cause intertrigo eczema The flaps should therefore be circular, and their combined lengths should be exactly equal to the diameter of the limb [1] Cutaneous nerves of the digits and thigh should be shortened slightly, but the main nerves of the limb should not be pulled down, crushed, ligated or injected with alcohol.[2] The formation of a nerve bulb cannot be avoided,[3] and its best situation is near the end of the stump where there will be no pressure Every bleeding vessel should be ligated so that hæmostasis is complete and drainage unnecessary. Stripping of periosteum or cutting of periosteal flaps is of no value, bone spurs form only on the posterior aspect of the femur and they give rise to no symptoms. The stump should be left at rest for two or three weeks, because early handling increases the tendency to development of the symptoms of phantom limb Firm bandaging is needed to promote shrinkage of muscles, and fitting of the artificial limb should be possible within two or three months of amputation

Indications for late amputation—Toxæmia from spreading infection, or from dangerous secondary hæmorrhage, gangrene from vascular thrombosis, or massive gas gangrene, may sometimes demand late amputation. Infection is then established in deep tissues and has spread proximally along lymphatics far beyond the limits of the wound Even when

[1] Ministry of Pensions "Artificial Limbs and Relation to Amputation" London H M Stationery Office, 1939
[2] Verrall, J "Amputation Stumps and Artificial Limbs" *Brit med J*, Jan 13, 1940, 62
[3] Page, Max 'Amputations under War Conditions" *Brit med J*, July 8, 1939, 77

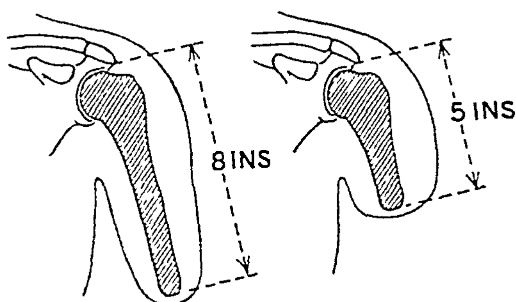


Fig 490

Optimal and minimal lengths of upper arm amputation stump

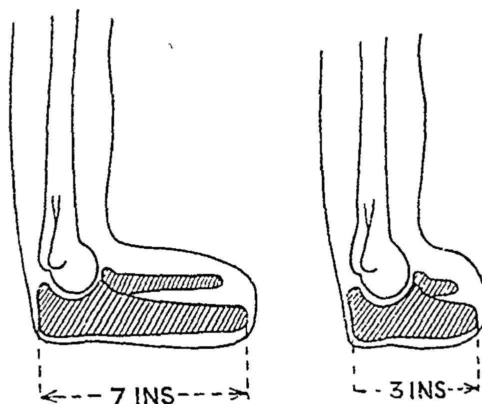


Fig. 491

Optimal and minimal lengths of forearm amputation stump

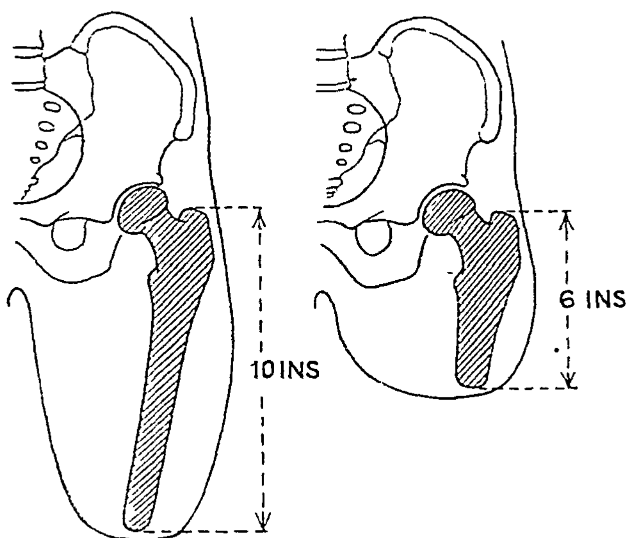


Fig 492

Optimal and minimal lengths of thigh amputation stump

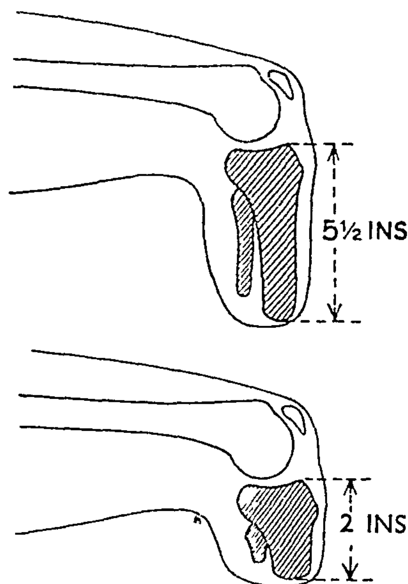


Fig 493

Optimal and minimal lengths of below knee amputation stump

Over 41,000 amputations were performed in this country during World War I Since then, tremendous strides have been made in the surgery of wounds and compound fractures, and amputation has become increasingly rare For example, in one large series of casualties treated in R A F Base Hospitals in World War II, the incidence of amputation for spreading infection, gas gangrene, hæmorrhage and other complications was as low as 0 1 per cent despite a high proportion of severely infected and grossly contaminated wounds and compound fractures

For details of the technique and management of amputation the reader is referred to an excellent monograph, *Amputations and Artificial Limbs*, by Langdale Kelham and George Perkins, Oxford War Manuals, London, 1942, and to the publication of the British Ministry of Pensions (M P M 414, 1950) entitled *Rehabilitation following Amputation*

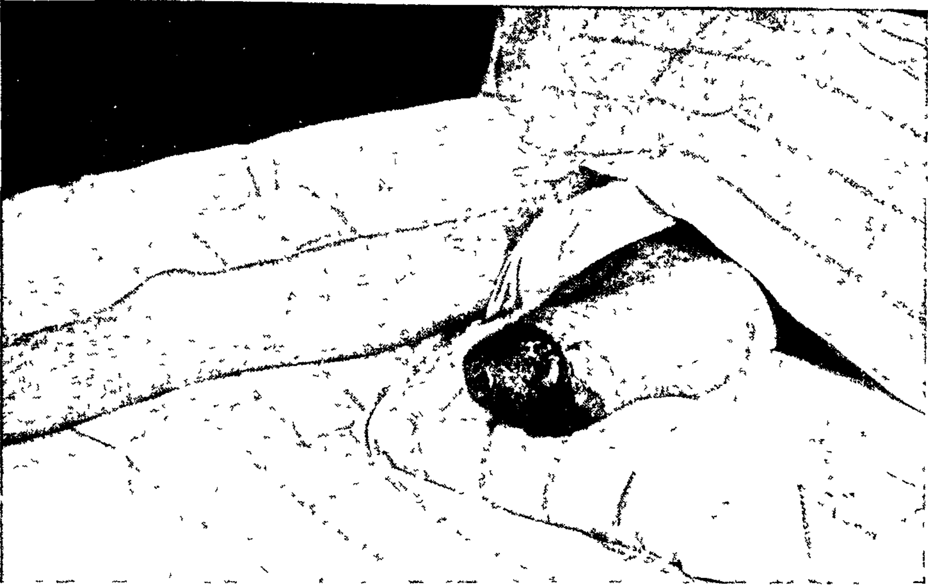


Fig. 494

Guillotine amputation left for ten days without skin traction, showing the rapid development of conical stump due to retraction of thigh muscles and skin, causing unnecessary shortening of the stump

Fig. 495

Method of application of skin traction with balanced weights, to prevent retraction of the skin and muscles after guillotine amputation (Note that the pulley should be at a slightly lower level and the stump should not be supported on a pillow, otherwise there is danger of flexion deformity of the stump.)

amputation is performed many inches above the level of injury, and when the wound is screened completely and the skin is prepared thoroughly, infection spreading along lymphatic channels cannot be avoided. Primary skin suture is unsafe

Technique of late amputation—Three choices are available 1) flap amputation with delayed skin suture after several days, 2) circular or sleeve amputation at a low level, with formal flap amputation at the site of election after two or three weeks, 3) guillotine amputation

Flap amputation with delayed suture—This is the operation of choice if the amputation must be performed at or above the site of election and if there is viable skin below that level (as in the case of gas gangrene). Flaps are cut but not sutured Mattress silkworm sutures may be put through the skin-flap margins, protected with rubber tubing or buttons, in order to apply light traction After several days, when the danger of spreading infection and gas gangrene is over, the flaps are sutured

Circular or sleeve amputation—A circular or sleeve amputation can be performed rapidly, with minimal exposure of tissues and with perfect drainage At the lowest level of safety—usually much below that of final amputation at the site of election—the skin is divided in one plane by a single sweep of the knife The muscles are then divided by a second sweep of the knife at a slightly higher level Finally the bone is sawn through, at a still higher level It is to be emphasised that this operation is not a guillotine division of all tissues of the limb at the same level Moreover, even with a true circular or sleeve amputation, skin traction should be applied within twenty-four hours The muscles of the thigh and leg are powerful and long bellied, and continued retraction of muscles and skin produces a conical stump with projecting bare bone, there is a wide area of granulation with unnecessary shortening of the stump (Fig 494) Traction is essential Vaseline gauze should be applied and the no-dressings technique used, so that unnecessary pain and fluid loss are avoided A formal flap amputation is then performed at the site of election after several weeks.

Guillotine amputation—The only indication for a true guillotine amputation is the exceptional case in which amputation must be performed at or above the site of election and there is no viable skin below that level. If the limb is divided in a circular manner, and skin traction is applied at once and continued until the granulating surface heals with a terminal scar, the stump may be satisfactory even without reamputation, whereas a flap amputation, which would necessarily be two or three inches higher, would give too short a stump

CHAPTER XIV

TRANSPLANTATION OF BONE

Few subjects have commanded a greater share of surgical literature than the growth and repair of bone, if we include only the surgical journals that have gained general recognition, a total of many more than five thousand papers have been published. The first of them occupied only two pages of the *Philosophical Transactions of the Royal Society* in 1738 John Belchier of London, a young surgeon on the staff of Guy's Hospital, had dined in the home of a calico-printer and noticed unusual redness in the bone of a leg of pork Learning that his host added the waste of printer's madder to pig-food, he experimented on fowls and published 'An Account of the Bone of Animals being changed to a Red Colour by Aliment only "[1] It was left to the remarkable Frenchman, Duhamel—a country gentleman and lawyer but not a doctor—to make the important discovery that *growing* bone alone was stained with dyes[4] By feeding madder alternately with ordinary food he produced concentric rings of red and white. and displayed the subperiosteal deposition of bone Growth in length was demonstrated by John Hunter,[5] who implanted lead-shot in the tibial shafts of chickens and showed that the pellets did not grow away from each other but that they became increasingly distant from the epiphyseal ends—which, as he had learned already from the teaching of his brother William, had an unusually rich supply of blood in the *circulus vasculosus et epiphyseos*.



FIG 496

John Hunter (1728-93)

From a portrait in the Royal College of Surgeons of England, reproduced in the *Journal of Bone and Joint Surgery*, 1948

The deductions of John Hunter—John Hunter also demonstrated that separated fragments of bone could survive and grow " Adhesion of detached splinters takes place not only in those which are attached to the soft parts but in those which are entirely loose " In concluding that " these pieces must retain the living principle " he made an accurate though quite unconscious forecast of the possibilities of bone transplantation Perhaps the most important deduction made by Hunter was that the shape of bone is determined by constant remodelling He examined the neck of the femur and the angle of the mandible, and satisfied himself that the changes in shape could not be explained by deposition of bone alone. " The remote causes of absorption of whole and living parts implies the existence of two conditions, the first of which is a consciousness in the part to be absorbed



Fig 497

The Pioneers of Bone Grafting

Syme Ollier	Duhamel Belchier Macewen	Goodsir Albee

of the unfitness or impossibility of remaining under such circumstances, whatever they may be, and therefore they become ready for removal and submit to it with ease. And the second is the consciousness of the absorbents of such a state in the parts. Both of them concurring they have nothing to do but to set to work" This exposition of bone resorption was made at a time when there was no microscope, no knowledge of cellular structure, and still less understanding of the function of osteoclasts and osteoblasts, yet Hunter's words were still no more than echoed two hundred years later in a recent monograph[19] "Bone looks so dead that it is easy to treat it as if it really were dead, as if it were no more than a block of marble carved with delicate artistry to fit the structures around it and give them support, and yet it is so very alive and forever busy shaping itself in adaptation to constantly changing demands."

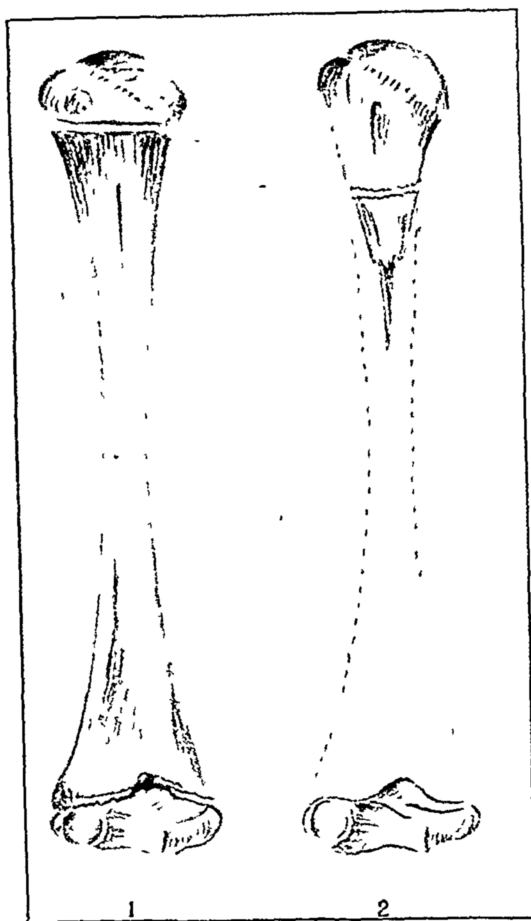
Function of the periosteum—The function of the periosteum was debated for nearly a century. Thirty-eight years after the death of John Hunter in London, James Syme[6] recorded his experiments in Edinburgh He removed part of the radial shafts of both forelimbs of a dog, preserving the periosteum on one side and destroying it on the other When the periosteum was saved there was new bone formation, when it was removed there was a gap In another dog he raised periosteum from a segment of bone and placed tinfoil beneath it; a layer of new bone was formed on the surface of the tinfoil Next he excised the periosteum and wrapped tinfoil round the denuded shaft; new bone was not laid down. He was satisfied as to the osteogenetic function of periosteum. Syme was then the acknowledged leader of surgical thought, and little was heard—and to this day little is remembered—of the work of his pupil and dresser John Goodsir, who became curator of the Museum of the Royal College of Surgeons of Edinburgh Goodsir[7] disclosed the fallacy of Syme's experiments By microscopic examination he showed that the raised flaps of periosteum included flakes of living bone Indeed it was Goodsir who discovered and described "bone cells" In the same year, Flourens in Paris repeated the experiments of Syme and made the same mistakes Even as late as 1857 Louis Ollier[8] of Lyons, when he was experimenting with the transplantation of rabbits' bone for the treatment of human un-united fractures, was convinced that periosteum was "the maternal tissue of bone"

The first successful bone transplantation—In 1887 Macewen in Glasgow removed the entire diaphysis of the humerus of a three-year old infant for persistent osteomyelitis[12] Three years later he was asked to amputate the

Some of the Classics of Bone Grafting

1 Belchier, J "An Account of the Bone of Animals being changed to a Red Colour by Aliment only" *Philosoph trans roy Soc* London, 1738, **39**, 287
2 Duhamel, quoted by Keith, 1919
3 Keith, Arthur "Menders of the Maimed" London Henry Frowde, Hodder and Stoughton Oxford University Press, 1919
4 Keith, Arthur "Bone Growth and Bone Repair" *Brit J Surg*, 1917 **5**, 685
5 Hunter, J "Works of John Hunter" London Longman, Rees, Orme, Brown, Green and Longman, 1835 37
6 Syme, J "Treatise on Excision of Diseased Joints" Edinburgh A Black, 1831
7 Goodsir, J "Anatomical Memoirs of John Goodsir" Vol 2, ed by Wm Turner Edinburgh A C Black, 1868
8 Ollier, L "Traite Experimental et Clinique de la Regeneration des Os et de la Production Artificielle du Tissu Osseux" Paris P Mason et Fils, 1867
9 Barth *Arch klin Chir*, 1908, **86**, 859
10 Axhausen, G *Arch klin Chir*, 1908, **88**, 23
11 Streissler "Der gegenwartige stand unserer klinische Erfahrungen uber die Transplantation lebenden menschlichen knochnens" *Brun's Beitr klin Chir Tubinger*, 1911, **71**, 1 (With bibliography on bone transplantation up to 1909)
12 Macewen, W "The Growth of Bone Observations on Osteogenesis An Experimental Inquiry into the Development and Reproduction of Diaphyseal Bone" Glasgow James Maclehose & Sons, 1912

flail and useless limb Instead, he implanted a number of bone wedges excised during corrective osteotomies from six other patients This was the first recorded example of successful homogenous bone grafting The transplanted bone regenerated and the rebuilt humerus measured six inches



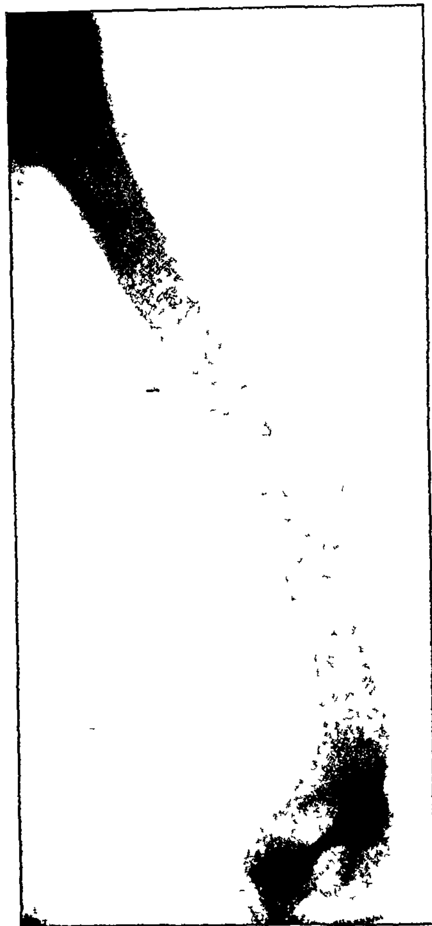


Fig 498

From Macewen's "The Growth of Bone," pages 177 and 189.

in length, more than two-thirds of it being from transplanted bone. Twenty years later the young man had a humerus eleven inches long, three inches shorter than the opposite humerus but with function so good that he earned his living in heavy manual work (Fig 498)

Some of the Classics of Bone Grafting

[13] Lane, W A "Fractures, Cleft Palate, etc" London The Medical Publishing Co Ltd, 1900
[14] Lane, W A "The Operative Treatment of Fractures" London The Medical Publishing Co Ltd 1914
[15] Groves, E W H "On Modern Methods of Treating Fractures" Bristol John Wright & Sons Ltd, 1st ed 1916, 2nd ed 1921
[16] Groves, E W H "Un-united Fractures with Special Reference to Gunshot Injuries and the Use of Bone Grafting" *Brit J Surg*, 1918, 6, 203
[17] Albee, F H "Bone Graft Surgery" New York D Appleton-Century Co, 1915, 1940
[18] Albee, F H "Orthopædic and Reconstructive Surgery" Philadelphia W B Saunders & Co, 1919
[19] Weinmann, J P, and Sicher, H "Bone and Bones" St Louis The C V Mosby Co, 1947
[20] Henderson, M S "The Massive Bone Graft in Un-united Fractures" *J Amer med Ass*, 1936, **107**, 1104
[21] Campbell, W C "Operative Orthopædics" London Henry Kimpton 1938
[22] Boyd, H B "The Treatment of Difficult and Unusual Non-unions with Special Reference to the Bridging of Defects" *J Bone Joint Surg*, 1943, **25**, 535
[23] Meekison, D M "The Treatment of Delayed Union or Non-union of Fractures by means of Massive Onlay Grafts fixed with Vitallium Screws" *J Bone Joint Surg*, 1945, **27**, 383

During the next twenty years many reports were published by the two German surgeons Barth and Axhausen, recounting their detailed and characteristically thorough experiments. Barth[9] concluded that the greater part of a bone graft underwent necrosis and replacement, that it was slow and indolent in growth, and that success depended largely on intimate contact of the graft with living vascular bone. Axhausen[10] showed that all cell spaces in the compact bone of a graft were empty but that there was cellular proliferation beneath the periosteum, bone formation from the marrow wherever it was in contact with living tissue and replacement of dead compact bone by new tissue invading it along channels made by young blood vessels.

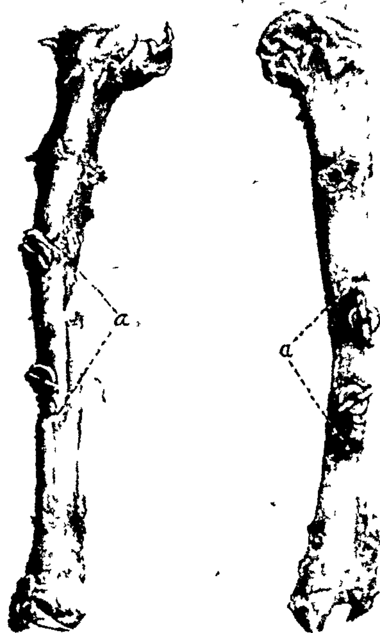


Fig 499

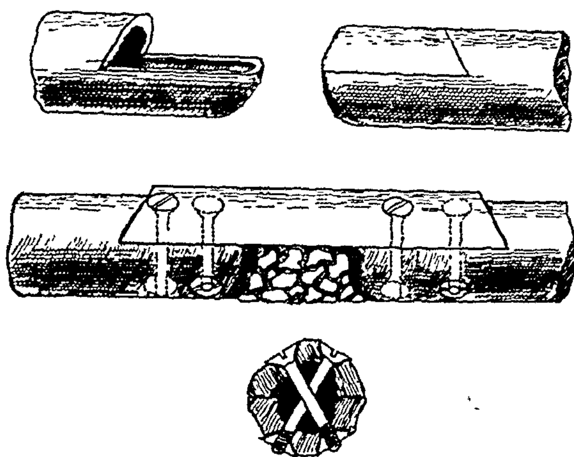


Fig 185—Half cylinder graft fixed by bolts In the upper figure the bone ends are shown cut for the reception of the graft In the middle figure the graft is seen bolted in place the pieces of bone cut from the original fragments being divided up and placed in the gap The lower figure shows the junction of the graft and the bone in transverse section

Fig 500

Massive onlay grafting with screw fixation, or with bolts and nuts, was described in England more than thirty years ago by Hey Groves These illustrations are from his lectures in the Royal College of Surgeons of England in 1916

Macewen's "Observations on Osteogenesis" was published in 1912[12] His critical and brilliantly simple experiments convinced him that Hunter and Goodsir had been right and that bone was regenerated and grew from the cavities of dead bone cells and from enlarged Haversian systems, that periosteal flaps produced bone only when they included bone cells; and that transplanted bone produced new bone independently of the periosteum Our knowledge of growth and repair, and of the behaviour of transplanted bone, was thus brought almost to where it stands to-day

Onlay and inlay grafting—Most earlier studies had been experimental and it was not until a technique of aseptic surgery had been introduced by Arbuthnot Lane that human fractures were operated upon with frequency Even the pioneer work of Lane, first published before 1900,[13 14] and of Hey Groves a few years later,[15 16] had limited success because it was not known that metal screws used for internal fixation must be electrolytically inert For this reason too the onlay grafts devised by Hey Groves often failed (Figs 499-500), and more uniform success was achieved by Albee of New York[17 18] whose technique depended upon a mechanically accurate fit

of the graft to its bed. The inlay graft cut with power-driven saws with "glass stopper" precision, was the basis of bone-grafting operations for the next decade, and surgeons who lived through it gained much in the experience of skilled carpentry in bone surgery Nevertheless inlay grafts



Fig 501



Fig. 502

Final development of the technique of massive onlay grafting had to await the discovery of biologically inert metals before it could be developed by Henderson, Campbell and Boyd

were necessarily slender—their width could not be much greater than half that of the bones into which they were inserted When inert alloys became available, stronger and wider grafts could be screwed on to the bone with a diameter at least as great as that of the host bone. Much then depended on the strength of the graft as a means of internal fixation of fractures, quite apart from any vital capacity it might have, and the treatment of un-united fractures by massive onlay-grafting with

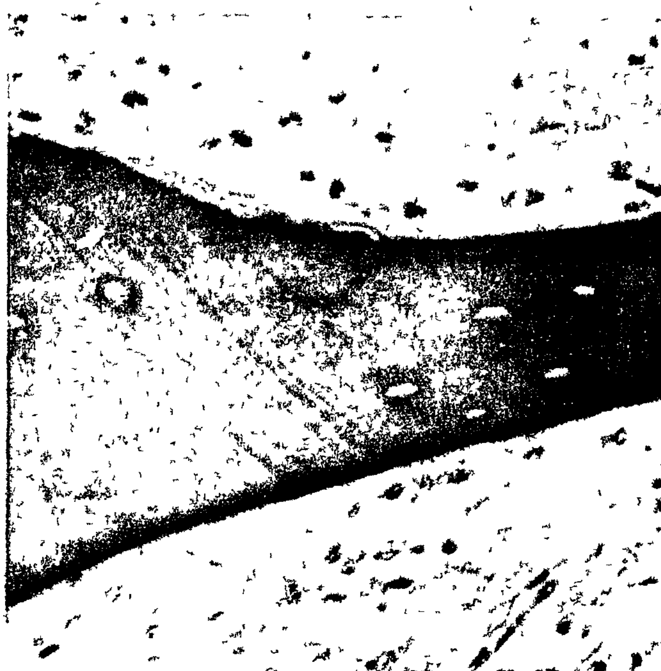


Fig. 503



Fig 504

The trabeculæ of transplanted bone die whether the bone is compact or cancellous Every cell space is empty (Fig 503) On the other hand surface cells survive and the speed of regeneration of cancellous iliac bone, as compared with compact tibial bone, depends solely on the fact that the slender trabeculæ (Fig 504) can be resorbed more quickly by the living osteoclasts on their surface, and be replaced quickly by the living osteoblasts

screw fixation was developed by Henderson,[20] Campbell,[21] Boyd,[22] and others.

The fate of transplanted bone—The question whether transplanted bone plays a passive or an active part in the healing of un-united fractures gave rise to controversy no less vigorous than that which had centred on the function of periosteum in earlier years Did the graft live, or was it regenerated after death? Throughout his life Albee maintained that bone grafts lived and grew as the twigs of trees survived when they were grafted. He made the peremptory observation that "the question of whether a bone graft lives when properly placed has been answered positively in the affirmative during the past thirty years of the author's experience, both in the animal research laboratory and in the clinic, at the operating table and in the follow-up of over sixty thousand cases" But great as was his conviction, Albee was wrong A bone graft does not survive as does a grafted twig Exactly the opposite view was expressed with no less emphasis by Leriche and Policard[24] "In man a fragment of transplanted bone always dies The question of death of a transplant should be considered solved It is thirty-two years since Barth conclusively showed it It is merely a waste of time still to seek to verify facts so well demonstrated The transplant has served only as a guide and a furnisher of calcium" This view was supported by Greig,[25] who reaffirmed that "transplanted bone never grows"



FIG 505

Showing resorption of dead trabecula by multinucleated osteoclasts on the right, and its replacement by new bone laid down by osteoblasts on the left This section is actually from a specimen of avascular necrotic bone in a fracture, but the histology is of course the same in the avascular bone of a transplantation

The truth was established finally by the work of Hey-Groves, Gallie Phemister, Stuck, Ghormley, Haas and others The greater part of the transplant does indeed die, all bone cell spaces are empty and the trabeculæ of bone, whether compact or cancellous, must be replaced (Fig 503) But cells on the surface survive, subperiosteal cells, endosteal cells and cells of the marrow proliferate, within a few days of transplantation osteoclastic activity is observed on one side of every dead trabeculum, while at the same time new bone is laid down by osteoblasts on the other (Fig. 505) The delay of regeneration of a compact graft depends only on the massive area of dead bone that must be removed through widening of the Haversian canals and progressive osteoclastic resorption The more trabecular the structure of a graft the more quickly is it replaced by living bone (Fig 504), the more compact it is, the more slow is its replacement.

Os purum and os novum—The earlier view that bone grafts served only as dead and inert scaffolding seemed to gain support from the fact that operations often succeeded when a graft was dropped accidentally to the floor of the operating theatre and was boiled before insertion Orell of Sweden[32] even advocated the routine use of " os purum," which was cadaveric human bone or animal bone treated by extraction of its proteins, boiling, and preservation in spirit The fact is, of course, that any un-united fracture will unite if the sclerosed surfaces are freshened, brought into apposition, and immobilised long enough , and that even dead bone used as an internal splint has architectural merit when it is compared with the structure of a metal plate But that is not to say that the trabecular structure of bone does not gain by having upon it living cells which at once initiate resorption of dead trabeculæ and replacement by living bone Almost as if relenting his advocacy of " os purum " Orell went on to describe " os novum," which was dead bone placed beneath the periosteum of a living tibia for several weeks before transplantation

Bone banks—Orell's purpose was to provide a store of bone He used dead bone from cadavers and animals , but Inclan[33] recognised that living bone was often removed in human operations and that it was wasted If such bone was preserved at a low temperature in sterile jars of citrated blood, or Ringer's solution, it might be transplanted at a second operation From this beginning the bone bank was developed It quickly gained popularity in America where the possibility of survival by refrigeration had already been accepted in the case of other tissues[41-46] Healthy bone from amputated limbs, or bone removed in the course of other operations, was refrigerated and transplanted as a substitute for fresh autogenous bone Ordinary refrigeration has been used to store grafts for three weeks, but it is believed that by " deep-freeze " refrigeration at a temperature of minus 25° C a graft can be preserved indefinitely Banks of merthiolated bone have also been used[38-40]

The purpose of such banks is to provide a reserve which makes it unnecessary to cut grafts from normal bones Every surgeon is aware that the tibia from which massive grafts are cut may sometimes be slower in regaining its function than the injured limb itself, and that secondary fracture of a tibia from which even small grafts have been cut is not uncommon Moreover with extensive defects, particularly in children, it is sometimes difficult to find enough normal bone to transplant Many

The Fate of Transplanted Bone

24 Leriche, R , and Policard, A " The Normal and Pathological Physiology of Bone " Translated by S Moore and J A Key London Henry Kimpton, 1928
25 Greig, D M " Surgical Pathology of Bone " Edinburgh Oliver & Boyd, 1931
26 Gallie, W E " The Transplantation of Bone " *Brit med Jour* , 1931, 2, 840
27 Phemister, D B " The Fate of Transplanted Bone and Regenerative Power of its Various Constituents " *Surg Gynec Obstet* , 1914, 19, 303
28 Ghormley, R K , and Stuck, W G " Experimental Bone Transplantation with Special Reference to the Effects of ' Decalcification ' " *Arch Surg* , 1934, 28, 742
29 Ghormley, R K " Choice of Bone Graft Methods in Bone and Joint Surgery " *Ann Surg* , 1942, 115, 427.
30 Haas, S L " *Function in Relation to Transplantation of Bone* " *Arch Surg* , 1921, 3, 425
31 Haas, S L " Spontaneous Healing Inherent in Transplanted Bone " *J Bone Joint Surg* , 1922, 4, 209
32 Orell, S *Surg Gynec Obstet* , 1934, 59, 683 *J Bone Joint Surg* , 1937, 19, 873

Bone Banks

33 Inclan, A " The Use of Preserved Bone Graft in Orthopædic Surgery " *J Bone Joint Surg* , 1942, 24, 81
34 Bush, L D " The Use of Homogeneous Bone Grafts " *J Bone Joint Surg* , 1947, 29, 620
35 Campbell, W C " Operative Orthopædics " 2nd ed Speed, J S , and Smith, H St Louis The C V Mosby Co , 1949, 123
36 Wilson, P D " Experiences with the Bone Bank " *Ann Surg* , 1947, 126, 932
37 Wilson, P D , Robert Jones Lecture, Royal College of Surgeons of England, 1950

successful results of transplantation from bone banks have been reported In a recent lecture in London, Philip Wilson[37] recorded a follow-up study of 248 transplantations of refrigerated bone of which 85 per cent. were successful But it must be recalled that success no less than this can be gained from the use of dead and boiled bone It has already been pointed out that every fracture unites if the bone surfaces are refreshened and the fragments are immobilised Firm immobilisation can be gained from the use of dead bone boiled bone or even bone from a bank Inclan believes that refrigerated homografts act like fresh autogenous bone, and that the histological appearances " vary little or not at all from fresh bone " Nevertheless it is not yet certain that the one merit of immediate transplantation of autogenous bone, namely the survival of surface cells, applies also to grafts that have been preserved in bone banks

The advance of recent years, much more important and offering no less satisfactory a solution to the justifiable reluctance of surgeons in removing large parts of a normal tibia, is the recognition that whereas hitherto bone grafts were expected to serve in dual capacity—as a means of providing internal fixation by their mass, and as a contribution to osteogenesis by their vitality—those two functions can and should be distinguished Internal fixation is provided better by intramedullary nailing, and osteogenesis is promoted better by cancellous chip grafts cut from the ilium The modern tendency of bone grafting for un-united fractures is to rely less on the accurate carpentry that was needed for inlay grafting, less on the damaging removal of bone from a normal tibia, less on the method of screw fixation with its peril of secondary fracture and more on the practice of refreshening the bone ends, filling the gap with cancellous chips cut from the ilium and providing internal fixation by medullary nailing

TECHNIQUE OF CORTICAL BONE GRAFTING

Methods of transplantation of cortical bone in the treatment of delayed union and non-union of fractures include the inlay graft of Albee[47], the sliding inlay graft of Kelly[48] the diamond inlay graft of Gallie[49], the intramedullary peg graft of Hoglund[50] and Hey Groves[51] the massive onlay graft of Henderson[52] and Campbell[53] the massive sliding onlay graft of Gill[54] and the double onlay graft of Boyd[55]

Inlay graft—Albee was the exponent of inlay grafts cut from the normal tibia and transplanted to the injured bone A bed was cut across the fracture

Merthiolate Bone Banks

[38] Morgan, C, Jameson, W A, and Powell, H M "Merthiolate as a Preservative for Biological Products" *Amer J Immun*, 1933, 25, 121
[39] Powell, H M, Jameson, W A, and Jones, J G *J Immunol*, 1933, 24, 185
[40] O'Connor, G B "Merthiolate, a Tissue Preservative and Antiseptic" *Amer J Surg*, 1939, 45, 563

Cartilage Banks

[41] O'Connor, G B, and Pierce, G W "Refrigerated Cartilage Isografts" *Surg Gynec Obstet*, 1938, 67, 796
[42] Brown, J B, and De Mere "Establishing Preserved Cartilage Bank" *Plastic and Reconstr Surg*, 1948, 3, 283

Skin Banks

[43] Webster, T D "Refrigerated Skin Grafts" *Amer J Surg* 1944, 120, 431
[44] Matthews, D N ' Storage of Skin for Autogenous Grafts" *Lancet*, 1945, 1, 775
[45] Strumina, M M, and Hodge, C C 'Frozen Human Skin Grafts" *Amer J Surg*, 1945, 121, 860
[46] Baxter, H, and Eatin, M A "Experimental and Clinical Studies on Reduced Temperatures in Injury and Repair in Man Direct Effect of Cooling and Freezing on Various Elements of Human Skin" *Plastic and Reconstr Surg*, 1948, 3, 303

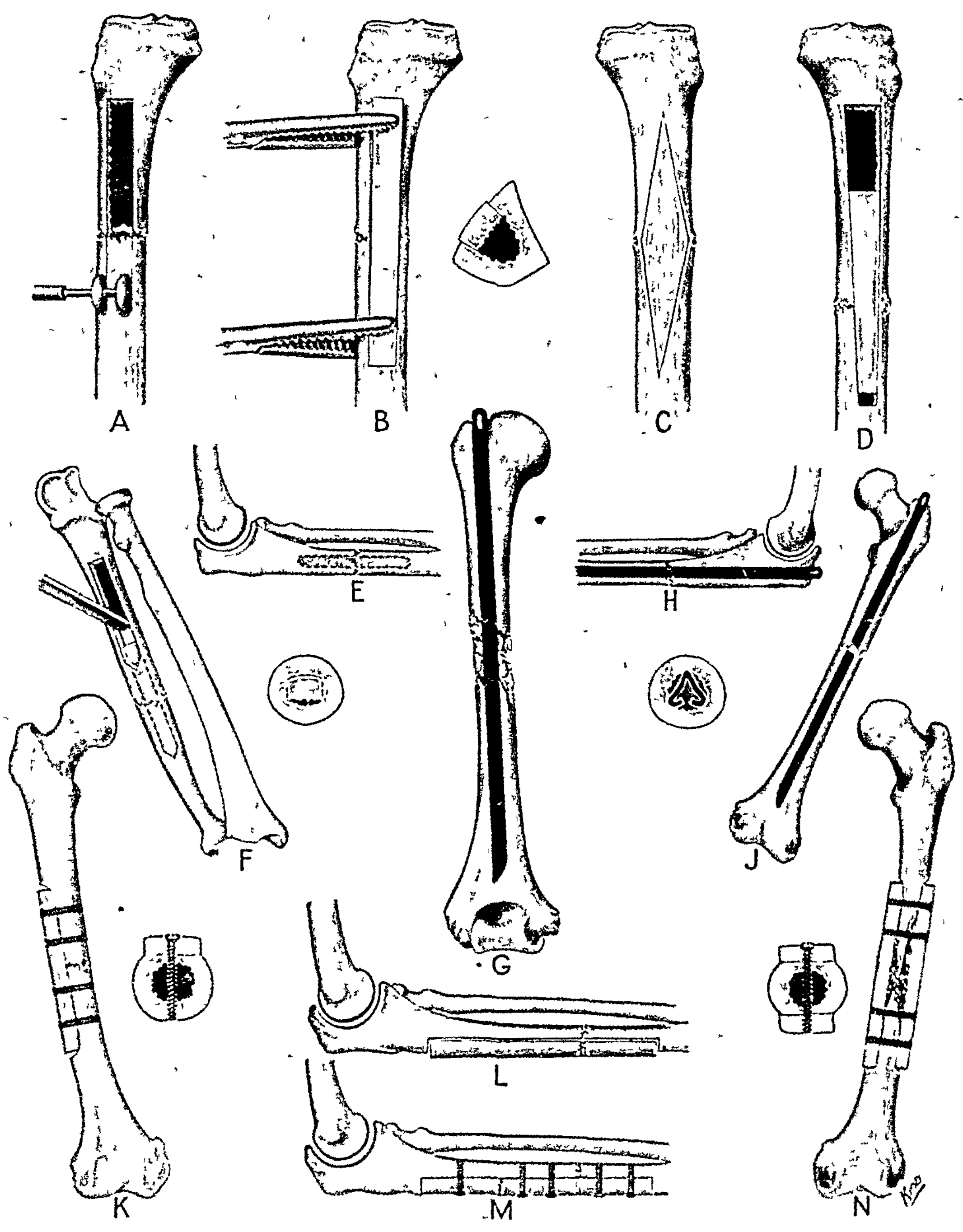


Fig. 506

METHODS OF BONE GRAFTING

Inlay, diamond inlay, sliding inlay, intramedullary peg, cancellous with intramedullary nail, onlay, sliding onlay, and double onlay grafts.

A, B—inlay grafting by the Albee technique, C—diamond inlay grafting; D—sliding inlay grafting, E—intramedullary peg grafting, F—sliding intramedullary peg grafting; G, H, J—intramedullary nailing with cancellous chip grafting, K—onlay grafting, L, M—sliding onlay grafting; N—double onlay grafting.

site with a twin saw, the interval between the saw-blades was then enlarged by the dimension of two saw-cuts so that the graft would equal in width the bed into which it was to be inserted. If the technique was accurate, the graft was sprung into its bed only with difficulty. Perfect fixation was achieved and the added fixation of screws, wire, or catgut was unnecessary. The inlay operation is still applicable to fractures of the femur, tibia and other large bones, but difficulties may arise in treating fractures of small bones such as the metacarpals, ulna and clavicle. The technique has now been largely replaced by other methods, but sometimes it may still be needed, as for example in the treatment of an un-united fracture of the lateral malleolus where onlay grafting would cause unsightly thickening (Figs 516-518).

Sliding inlay graft—An alternative type of inlay avoids the necessity for a second operative exposure by which the graft is cut from healthy bone. A graft cut from one fragment of the fractured bone is slid across the fracture-site into the other fragment. If the sliding graft is rectangular in shape it will be narrower than the bed by the width of two saw-cuts. A sliding graft should therefore be wedge-shaped (Fig. 506, D), and the exact angle of the wedge must be judged with precision[48] The technique has not a wide application because the fractured bone is often so porotic that a graft cut from one of the fragments has little strength. Moreover, the site of fracture may make it difficult to cut a graft that is long enough. Short grafts are useless. In most long bones the leverage at the site of fracture is such that a graft must measure at least four inches in length. Many failures have resulted from the determination to avoid operating on normal bones and the endeavour to cut a graft from one fragment of a fractured bone which is obviously too short for the purpose (Figs 537-538).

Diamond inlay graft—Gallie was the exponent of the diamond-shaped bed and graft. The greatest width of the graft was at the level of fracture where there was most sclerosis. An accurate template of the gap in the recipient bone was prepared, and placed on the host bone before the graft was cut; but it is obviously more difficult to cut a diamond-shaped graft accurately than to prepare a rectangular graft with a twin saw.

Intramedullary peg graft—The intramedullary insertion of a graft is the least satisfactory technique because if no more than the medulla is drilled the greater part of the sclerosed surface is left undisturbed, and the only source of new bone growth is plugged (Figs 511-512). Even if sclerosed bone is cut away, and the gap is filled with a cricket-bail graft pegged into each end by the technique of Hey Groves,[51] the results are still unsatisfactory because the graft is weak where it is dowelled. Moreover, an intramedullary peg controls only angulatory movement and offers little resistance to rotational strain. Sliding intramedullary grafts in which a piece of bone cut from one fragment is pegged across the fracture site (Fig. 506, F) have

Technique of Bone Grafting

47 Albee, F H "Bone Graft Surgery" New York D Appleton-Century Co, 1915, 1940
48 Kelly, R E "Wedge Principle in Fashioning a Tibial Bridge Graft" *Brit J Surg*, 1923, 10, 232.
49 Gallie, W E *Brit med J*, 1931, 2, 840
50 Hoglund, E J *Surg Gynec Obstet*, 1917, 24, 243
51 Groves, E W. H "On Modern Methods of Treating Fractures" Bristol 2nd ed, 1921, 269.
52 Henderson, M S *J Amer med Ass*, 1936, 107, 1104
53 Campbell, W C "Operative Orthopædics" London Henry Kimpton, 1938
54 Gill, A B *Surg Clin N. Amer*, 1932, 12, 1535
55 Boyd, H B. *J Bone Joint Surg*, 1943, 25, 535

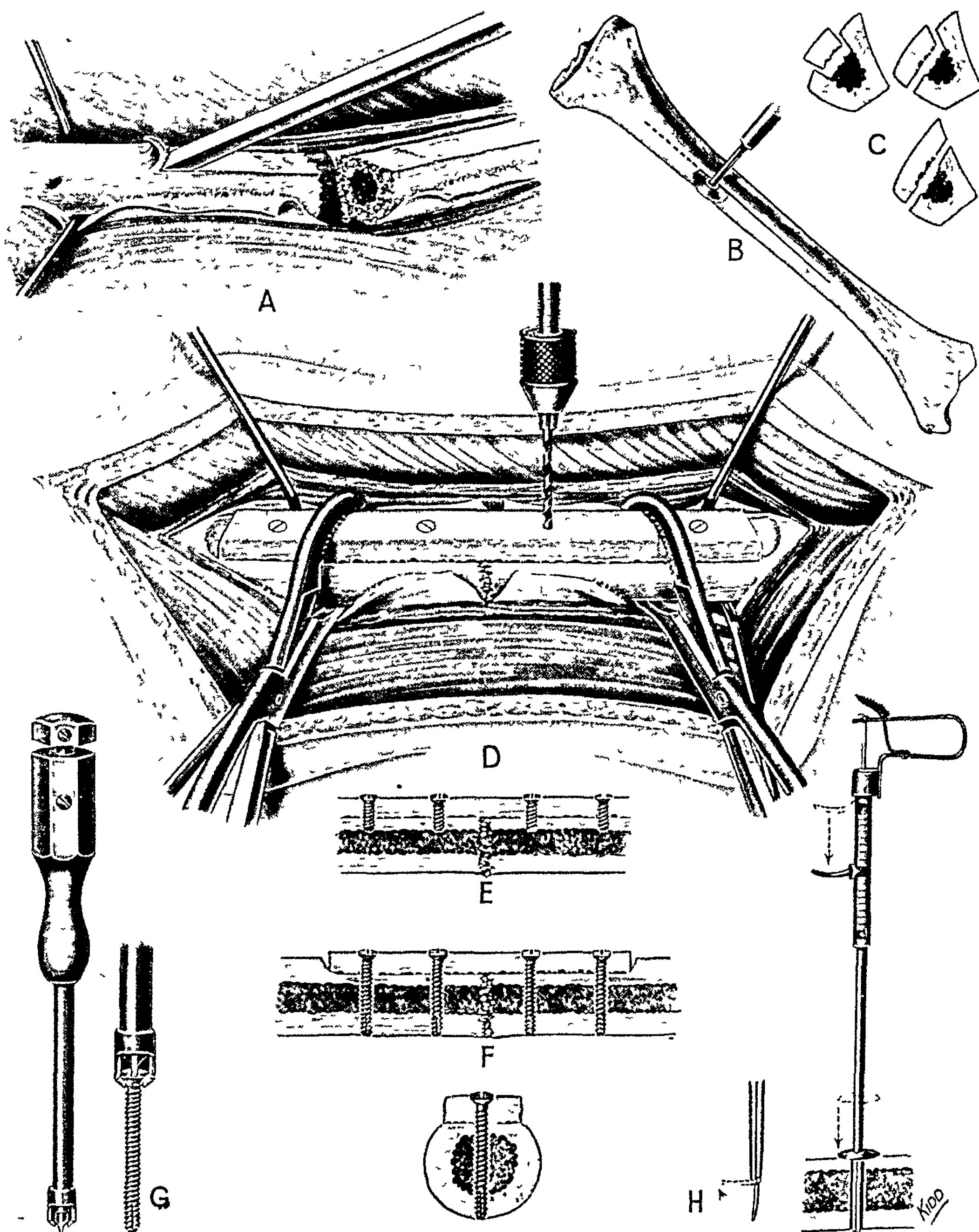


FIG. 507

Technique of massive onlay grafting of bone

The ends of the fractured bone are freshened and a bed is prepared on the surface of each fragment with a chisel (A). The graft cut from the tibia may include one or both cortical margins (B, C). The graft is fixed to the host bone by screws inserted after a track has been drilled (D) The screws must engage the opposite cortex (F) and the length should be measured accurately (H). A screw-holding driver is almost indispensable (G)

all the disadvantages of other intramedullary methods; the graft is not long enough, it plugs the medullary source of new bone; and the internal fixation is inadequate

Massive onlay graft—This is the technique described originally by Hey Groves and later adopted and popularised by Henderson, Campbell and Boyd[52,55] It has the merit that powerful fixation is secured between the graft and host bone over a wide area so that the endosteal surface of the graft is in firm contact with living cells of the host bone, and quick revascularisation and regeneration is assured (Figs 519-540) The fractured ends of the bones are exposed and freshened Intervening scar tissue is excised A bed is then cut on one side of the fractured bone so that a healthy vascular area is opened widely, and a flat surface is freshened to which the tibial graft can be applied The bed should not be cut too deeply; the more completely the parent bone is preserved the more soundly will fixation be secured by the onlay graft The bed should be cut with a chisel rather than a power-driven saw in order to avoid burning of the host bone. The graft from the healthy tibia should be at least as wide as the host, it may include one or even both cortical margins. While the graft is held securely with bone-holding forceps, holes are drilled and screws are inserted. The screws must engage the opposite cortex, and their exact length should be determined accurately by a screw-measuring device (Fig 507, H) There should usually be two screws on each side of the fracture

Massive sliding onlay graft—In fractures of the tibia it is sometimes possible to cut a massive onlay graft from one fragment and slide it across the site of the fracture, fixing it with screws exactly as in other onlay grafting procedures The technique was advocated by Gill[54] (Figs 529-530)

Twin onlay graft—On some occasions the host bone is so slender, thin and porotic that even with transfixing screws the fixation of an onlay graft is insecure This problem arises particularly in congenital pseudarthrosis of the tibia where the ends of the fragments are spindle-shaped and offer little possibility for the fixation of a single graft Boyd[55] showed that in these cases twin grafts could be applied with the fixation of a vice. The technique has merit also in the fractures of adults where there is wide separation between the fragments and severe porosis of the bones (see Figs 541-542 and compare with Figs 524-525)

TECHNIQUE OF CANCELLOUS BONE GRAFTING

The practice of filling the gaps of an un-united fracture with fragmented bone is not new—it was indeed the basis of the first successful grafting operation performed nearly seventy years ago by Macewen Nevertheless it is only within recent years that the special merit of cancellous bone has been recognised It began with Matti in 1932,[1] and gained impetus from the work of Rainsford Mowlem[2,3] and Abbott[4,5] who urged that the vitality of a cancellous graft with its quick regeneration was more important

[1] Matti, H "Uber freie Transplantation von Knochenspongiosa" *Arch Klin Chir*, 1932, 168, 236
[2] Mowlem, A R "Report on Eighty-five Cancellous Chip Grafts" *Lancet*, 1944, 2, 746
[3] Mowlem, A R "Cancellous Chip Grafts for the Restoration of Bone Defects" *Proc Roy Soc Med*, 1945, 38, 171
[4] Abbott, L C, Saunders J B, and Bost, F C "Arthrodesis of the Wrist with the Use of Grafts of Cancellous Bone" *J Bone Joint Surg* 1942 24, 883
[5] Abbott, L C "The Use of Iliac Bone in the Treatment of Un-united Fractures" *Amer Acad Orth Surg* Lectures on Reconstruction Surgery, Edwards Bros, Ann Arbor, 1944

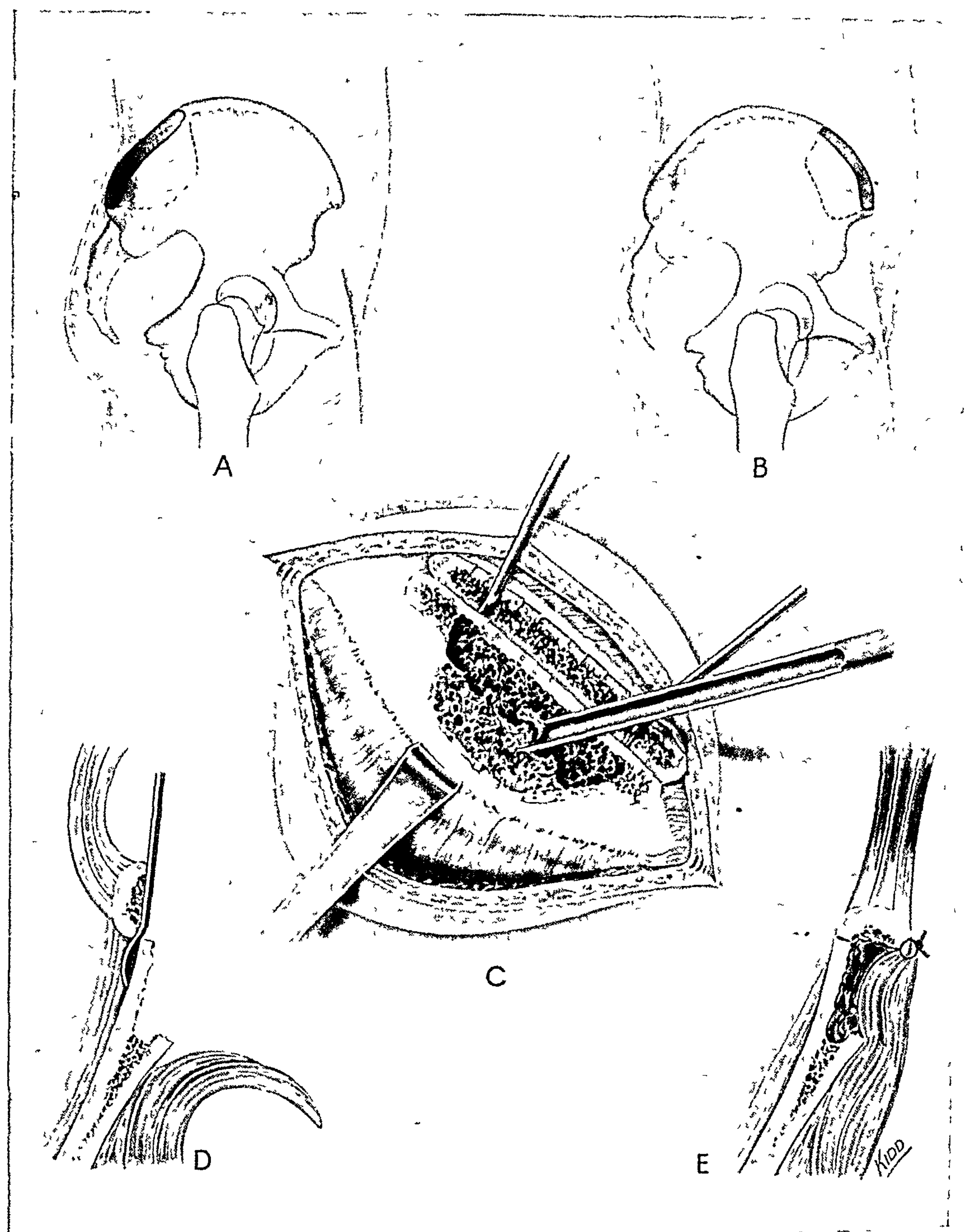


FIG 508

Cancellous chip grafting of bone

Cancellous chip grafts should usually be cut from the ilium in the region of the posterior superior spine and the crest (A) or from near the anterior superior spine (B) The crest is reflected with its attached muscles and the grafts are then cut with a gouge (C). After suture of the glutei to the abdominal muscles the iliac crest is reconstituted (E).

than the strength of a compact graft with its quality of immobilisation. Cortical grafts of compact bone had been expected to serve the dual function of providing internal fixation and promoting osteogenesis; but immobilisation can often be gained more securely by plates or medullary nails, and osteogenesis can be stimulated more certainly by cancellous bone. It is true that bone cells die in a cancellous graft exactly as they die in a compact graft, but dead cancellous bone can be resorbed and regenerated more rapidly than dead compact bone. Thus the tendency of the modern treatment of un-united fractures is to use cancellous transplants without the use of splints of compact bone; to use cortical bone only when no other method of internal fixation is available, and even when cortical bone must be used—whether it is from the patient's own tibia or is boiled cadaveric bone or bone from a bank—to supplement it with fragments of cancellous bone cut from the ilium. As a rule, the sclerosed ends of the un-united fracture are freshened and an intramedullary nail is inserted, the fracture site is then packed with iliac cancellous chips crushed into each other as tightly as possible.

The indolence of compact bone is still more important if un-united fractures are infected, or if they are potentially infected by reason of recent healing of wounds. Osteoclastic resorption of dead compact bone is seldom if ever complete when there is infection, and the residual fragment remains as a sequestrum. On the other hand cancellous bone with its thin trabecular structure offers little resistance to osteoclastic resorption; the dead trabeculæ are removed completely while at the same time new bone is laid down. When there is actual or potential infection, transplants of cancellous bone alone should be used. It is just as important to avoid using compact bone as it is to avoid using plates, screws, nails or other foreign bodies which serve as sequestra and cause persistent infection.

The best source of cancellous bone is the crest of the ilium in the region of the posterior superior spine, and to a lesser extent the anterior superior spine. The head of the tibia is less satisfactory because there is a high proportion of fat and marrow in the bone. A thin margin of the iliac crest is cut with an osteotome and reflected with the spinal and abdominal muscles still attached. The cortex of the dorsum ilii is then removed and this uncovers a rich supply of vascular cancellous bone (Fig. 508). It should be removed with a gouge without perforation of the inner table—one or two cases of internal hernia through complete defects of the ilium have been reported. When enough bone has been removed—and by enough we mean sufficient to pack the defect very tightly—the crest of the ilium is replaced and the attached muscles are sutured firmly to the glutei so that bleeding will be controlled.

Examples of the treatment of un-united fractures by cancellous bone alone are shown in Figures 558-572; and the application to arthrodesis of joints is seen in Figures 554-557. The use of cancellous chips to supplement tibial onlay grafts is demonstrated in Figures 541-542. The importance of using cancellous bone alone when there is infection, and the failure of compact grafts in these circumstances, is proved in Figures 560-564. The occasional case in which there is need to promote stability, and yet such danger of infection that tibial cortical bone must not be used and that slabs of iliac bone including its thin cortex are transplanted, is illustrated in Figures 555-556.

TREATMENT OF UN-UNITED FRACTURE BY SIMPLE REFRESHENING OF THE FRACTURED SURFACES AND IMMOBILISATION OF THE FRAGMENTS (FIGS. 509-510)



FIG. 509



FIG. 510

Ten years before this patient was first seen a supracondylar osteotomy of the femur had been performed by Sir Robert Jones for genu valgum, but the osteotomy had failed to unite. The fractured surfaces were refreshened and the limb was immobilised in a plaster spica for four months. The bone united soundly.

TREATMENT OF UN-UNITED FRACTURES BY INTRAMEDULLARY PEG BONE GRAFTING (FIGS. 511-512)



FIG 511



FIG 512

Intramedullary peg bone grafting was successful in this case, but the method is not recommended because such a graft does not really immobilise the fragments, and moreover, since it plugs the medulla, a solid intramedullary peg interferes with an important source of new bone growth.

UN-UNITED FRACTURE OF THE FEMORAL CONDYLES TREATED BY PEG GRAFTING (FIGS. 513-515)



FIG 513



FIG 514



FIG 515

Although peg grafting is not recommended for un-united shaft fractures it is sometimes needed for fractures near the ends of the bones—the neck of the femur, the medial malleolus or, as in this case, the femoral condyles There was no union between the two condyles or between either condyle and the shaft The surfaces were refreshed, both condyles were pegged to the shaft by tibial grafts, and the genu varum was corrected by osteotomy above the level of the un-united fractures The limb was immobilised in a plaster spica The fractures united soundly and 90 degrees of knee movement was regained

THE OCCASIONAL CASE WHEN INLAY BONE-GRAFTING IS STILL NEEDED (FIGS. 516–518)



Fig. 516



Fig. 517



Fig. 518

Inlay bone grafting has been replaced almost entirely by onlay bone grafting with screw fixation—or by cancellous chip grafting with medullary nail fixation—but there are still occasions when an inlay graft is better. In un-united fractures of the malleoli an onlay graft would thicken the ankle and cause disfigurement. In this rare example of an un-united fracture of the *lateral* malleolus union was secured without thickening of the bone by an inlay graft.

TREATMENT OF UN-UNITED FRACTURE OF THE SHAFT OF THE TIBIA BY ONLAY GRAFTING WITH SCREW FIXATION
(FIGS 519-520)



FIG 519

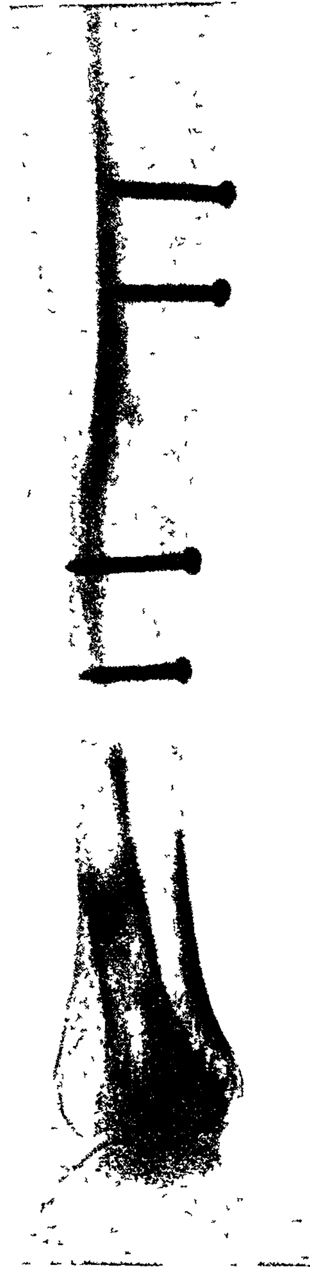


FIG. 520

Onlay grafting with vitallium or stainless steel screw fixation has almost entirely replaced inlay grafting even in larger bones such as the femur, tibia and humerus, because much more complete immobility can be secured by this technique This is a twelve-month-old un-united fracture of the shaft of the tibia (Fig 519) treated by an onlay graft cut from the opposite tibia and fixed with four vitallium screws (Fig 520)

UNUSUALLY LONG ONLAY GRAFT FOR DOUBLE FRACTURE OF THE TIBIAL SHAFT ASSOCIATED WITH OTHER INJURIES (FIGS. 521-526)

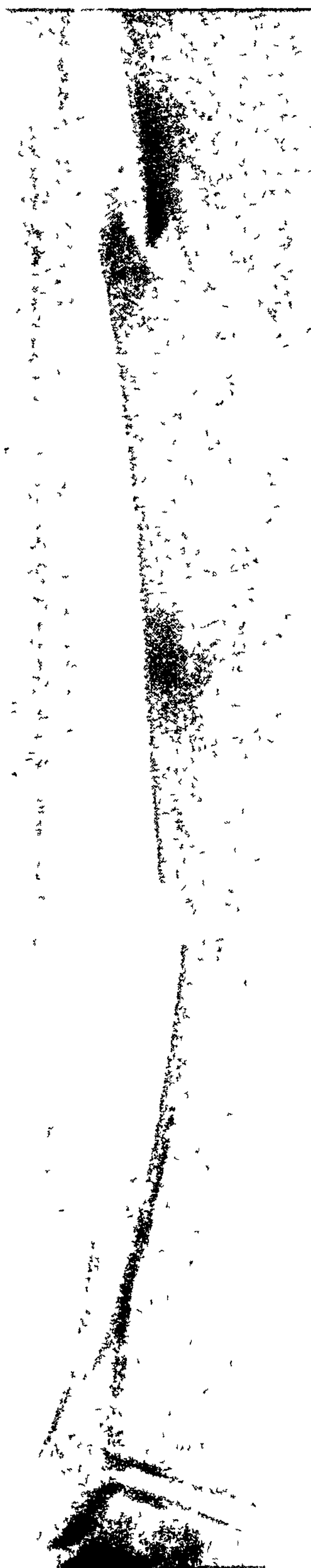


FIG 521



FIG 522

Double fracture of the shaft of the tibia with distraction of the fragments. See also Figures 523-526, and legend on opposite page

Fig 523



Fig. 524



Fig 525



Fig 526

Figs. 521-526

A Royal Air Force pilot sustained multiple injuries including a double fracture of the tibial shaft (Fig. 521) Attempted correction of alignment and length caused distraction of the distal fracture (Fig 522) and the treatment was made more difficult by a comminuted fracture of the femur of the same limb (Fig 524) The tibial fractures were supported in a below-knee plaster while the fractured femur was immobilised in traction in a Thomas' splint As soon as the femur was united a long graft cut from the opposite tibia was onlaid across both tibial fractures (Fig 523) There was also a comminuted fracture of the head of the radius which was excised (Figs 525-526)

(The operations were performed by Mr Armstrong, now of London, while in the Orthopædic Service of the Royal Air Force)

ONLAY GRAFTING FOR GAP FRACTURES (FIGS. 527-528)

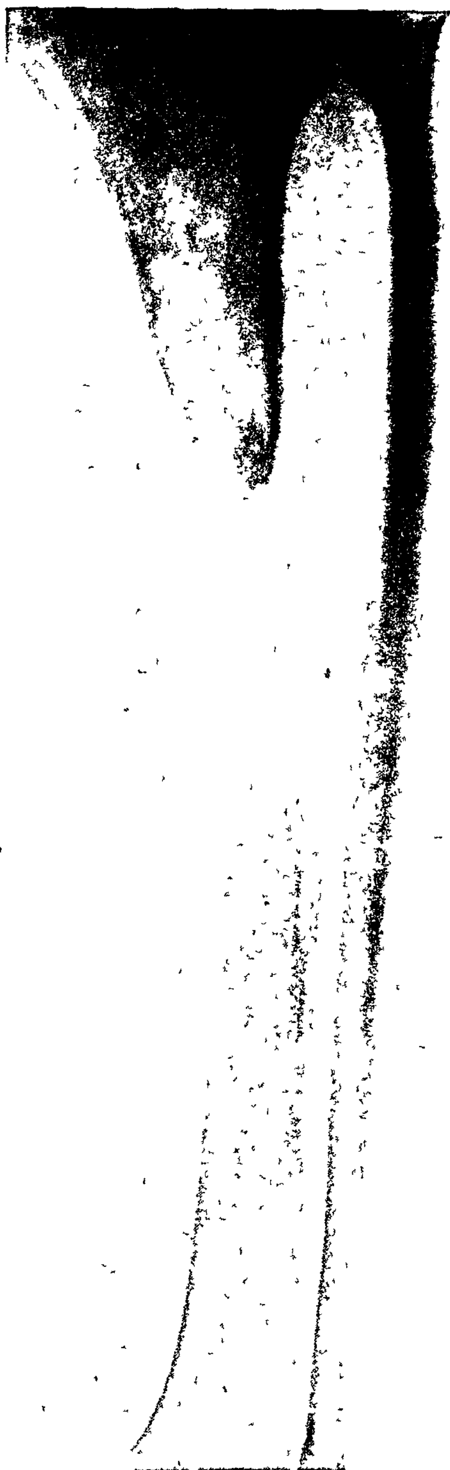


FIG 527



FIG 528

Un-united gap fracture of the shaft of the tibia from a shell wound (Fig 527) It was treated by an onlay graft from the opposite tibia, but a mistake was made in filling the gap with another whole-thickness graft placed end to end between the fragments of the host bone (Fig 528) Quicker union would have been gained if the gap had been filled with cancellous bone chips, or still better if a double onlay graft had been combined with the insertion of iliac chips (Compare Figs 541-542)

SLIDING ONLAY BONE GRAFT (FIGS. 529-530)



Fig 529 Fig. 530

For fractures of the middle and lower thirds of the shaft of the tibia an onlay graft may be slid down from the upper fragment by the technique described by Gill and advocated by Armstrong. The crest, together with part of the lateral and medial surfaces of the tibia, is cut from both proximal and distal fragments The longer piece of bone from the proximal fragment is displaced downwards and screwed in position across the site of fracture The smaller piece of bone from the distal fragment is used to fill the gap in the upper fragment and is fixed in position by one screw.

ONLAY GRAFT CUT FROM THE SAME BONE (FIGS. 531-534)

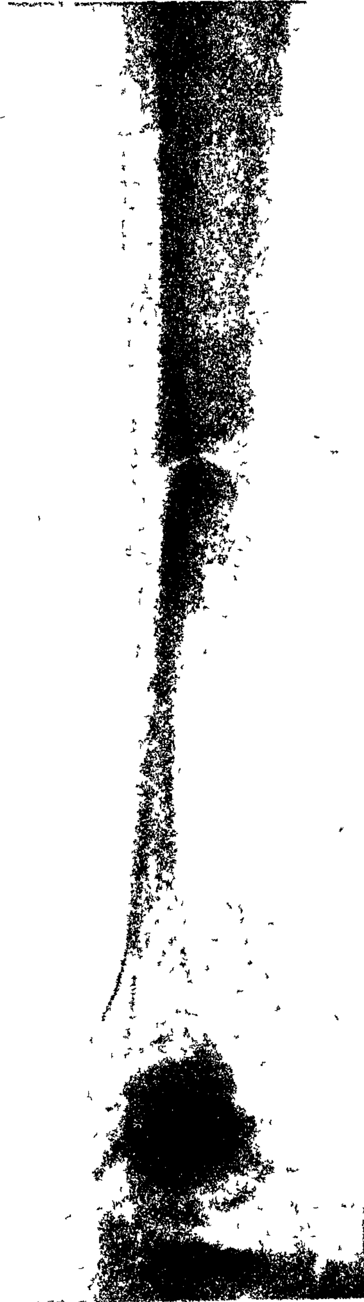


Fig. 531



Fig. 532

In onlay grafting there is obvious merit in avoiding the cutting of bone from a normal t and this is often possible. In many ununited fractures of the lower shaft of the tibia a graft ca cut from the upper shaft of the same bone (Fig. 532). The cause of non-union in this case avascularity of the distal fragment. When exposed at operation the distal fragment of the t unlike the proximal fragment, was quite bloodless, and microscopic section shows that th was infarcted (Fig. 541). Sound union was secured by onlay grafting.



FIG 533

Microphotograph of bone from the *proximal* fragment of the fractured tibia shown in Figure 531—it is alive



FIG 534

Microphotograph of bone from the *distal* fragment of the fractured tibia shown in Figure 531—it is dead The blood supply has been lost, and every bone cell space is empty, but endosteal and marrow cells, seen below, have survived and from them the bone regenerates (Refer to p 17)

ONLAY GRAFT CUT FROM THE SAME BONE BUT OF INSUFFICIENT LENGTH—COMPLICATED BY ARTERIO-VENOUS ANEURYSM (FIGS. 535-540)



Fig. 535



Fig 536



Fig 537



Fig 538

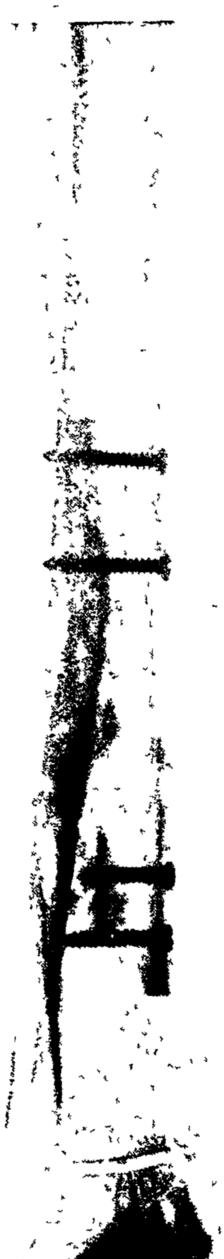


FIG. 539



FIG 540

The surgeon who referred this case had inserted a Steinman pin in the distal tibial fragment with the object of transfixing it and including the pin in the plaster to prevent recurrent bowing, he reported that shortly afterwards "the limb began to purr like a kitten" Perforation of the anterior tibial artery and veins had caused an arteriovenous aneurism The aneurism was excised and at the same time the un-united fracture was treated by an onlay graft cut from the same bone, but the graft was short and it failed (Figs 535-538) A longer and stouter graft cut from the opposite tibia was successful (Fig 539) As a rule, onlay grafts for the tibia are placed on the lateral muscular surface, but in this case it was placed on the anteromedial surface in order to control the outward bowing more easily The four screws were later removed (Fig 540)

DOUBLE ONLAY BONE GRAFTING FOR GAP FRACTURES PARTICULARLY WHEN THE BONE IS POROTIC (FIGS. 541-542)

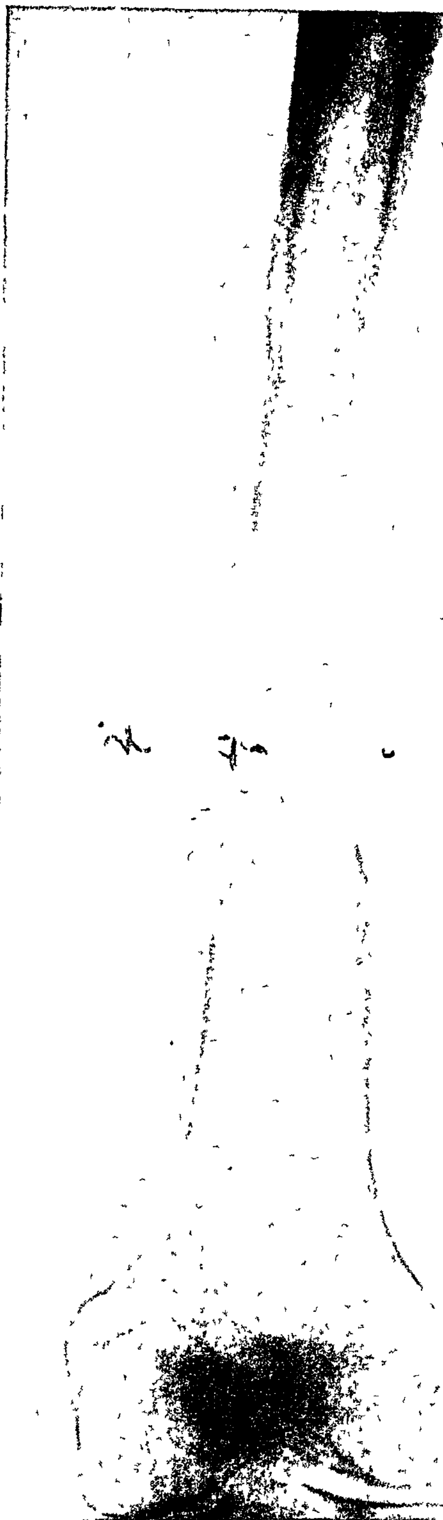


FIG 541

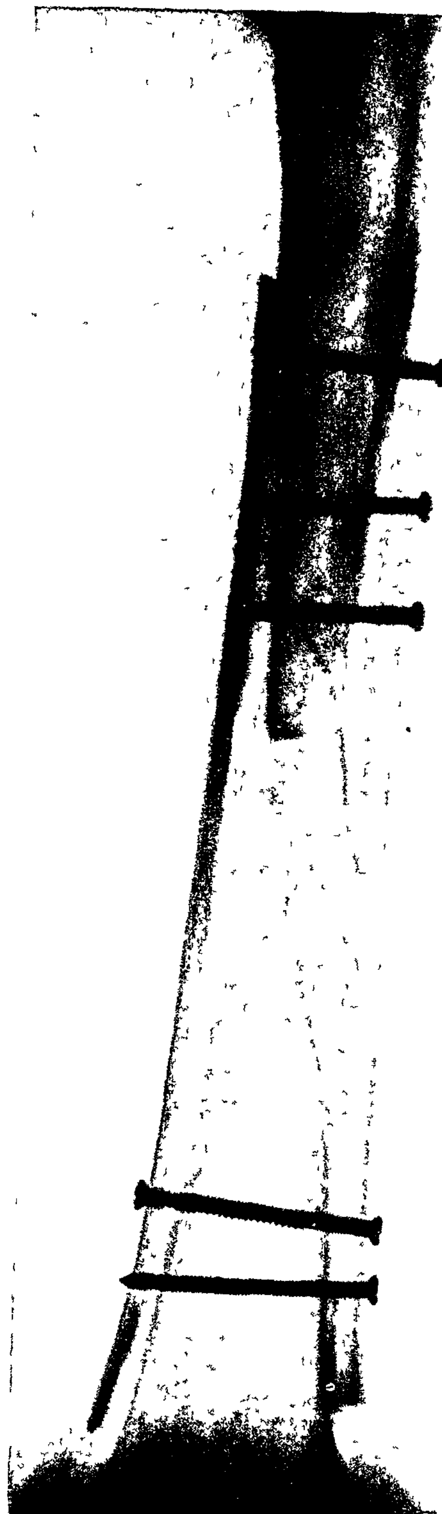


FIG 542

In this un-united gap fracture of the shaft of the femur the bone was so porotic that there would have been danger of failure from a single onlay, from rapid loosening of the screws A double onlay graft gives much greater fixation (Fig 542) The gap was filled with cancellous bone chips cut from the ilium, and the fracture united quickly (Compare Figs 527-528) *(The operation was performed by Mr A Butler, now of Montreal, when he was in the R A F. Orthopædic Service)*

DOUBLE ONLAY GRAFT FOR DIFFICULT UN-UNITED FRACTURE OF THE UPPER END OF THE ULNA (FIGS. 543-544)



FIG 543

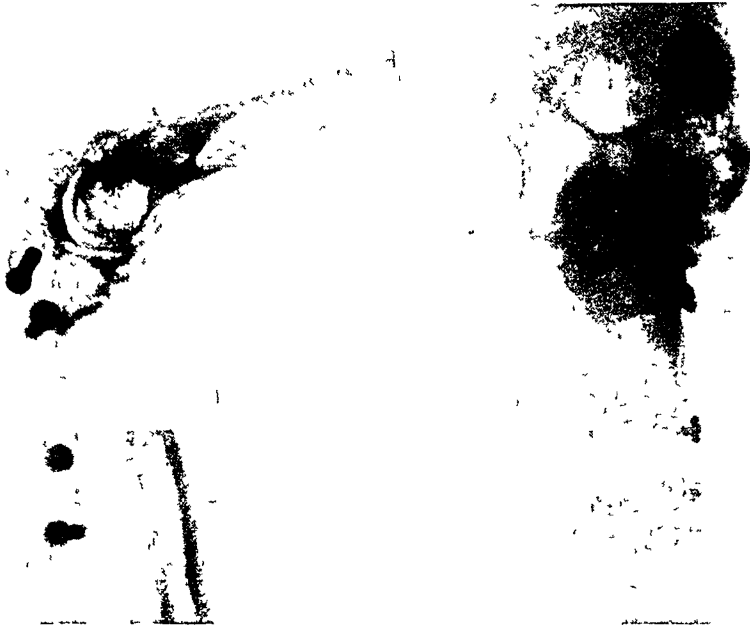


FIG 544

This was an open Monteggia fracture-dislocation in which a good deal of bone had been excised from the unusually high fracture of the ulna, the radial head had been excised but the shaft had displaced upwards to lie in contact with the capitellum (Fig 543) The problems were to secure union of the ulna, and to restore movement of the radius Union of the fractured ulna was gained by means of a double onlay graft; but, despite free excision of the upper end of the radius, and covering of the stump with muscle flaps (Fig 544), new bone formation limited radio-ulnar movement The elbow joint was strong, and half the normal range of movement was regained

COMBINED ONLAY AND PEG GRAFT WITH THE FIXATION OF A SINGLE SCREW FOR UN-UNITED FRACTURES OF THE METACARPAL (FIGS. 545-547)



FIG. 545



FIG. 546



FIG 547

A Czech pilot sustained a gunshot wound which destroyed the proximal part of the index metacarpal Lack of stability of the stump of the metacarpal made it impossible for him to mobilise the metacarpo-phalangeal joint which was stiff in hyperextension. One surgeon said that no operation should be done because movement of the finger depended on the pseudarthrosis! We have heard the same observation in relation to stiffness of the elbow joint associated with un-united supracondylar fractures If stability is achieved by union of the fracture, the joint can then be mobilised by active exercise, it is quite fallacious to accept pseudarthrosis near a joint with the object of preserving movement A tibial graft was pegged into the trapezoid and fixed by one screw to the metacarpal fragment after it had been step-cut to receive it The metacarpo-phalangeal joint had already been manipulated into the flexed position and for a few weeks it was held in this position in plaster The graft became incorporated and the fracture united, *normal* movement of the joint was regained.

CASE TO ILLUSTRATE THE DISADVANTAGE OF THE SCREW FIXATION NEEDED FOR METAL PLATES AND ONLAY BONE GRAFTS (FIGS. 548-549)



Fig 548



Fig 549

This was a case of congenital dislocation of the hip joint in which the roof of the acetabulum had been reconstructed, nevertheless subluxation recurred because there was 90 degrees of anteversion of the femoral neck The femoral neck-shaft relationship was restored by a rotation osteotomy, the fragments being fixed by a plate and four screws The osteotomy united, the anteversion was corrected, and the femoral head lay normally in the acetabulum All was well But two years later, in consequence of a stumble, the femoral shaft was fractured through the site of the lowest screw Screw fixation of both cortices of a bone, whether for the fixation of a plate or of an onlay graft, is an occasional but unfortunate necessity The technique of bone grafting with screw fixation is being replaced rapidly by cancellous chip grafting with intramedullary nail fixation

PERSISTENT NON-UNION OF THE FOREARM. GREATLY SHORTENED AFTER REPEATED TREATMENT. EXCELLENT FUNCTION AND SOME LENGTH RESTORED BY ONLAY GRAFTING



Fig 550



Fig. 551

Many surgeons in South Africa, Australia, America and England had tried to secure union of this fracture of the forearm Every one of the many operations that had been performed had been preceded by "freshening the bone ends"—and by the time the patient



FIG. 552

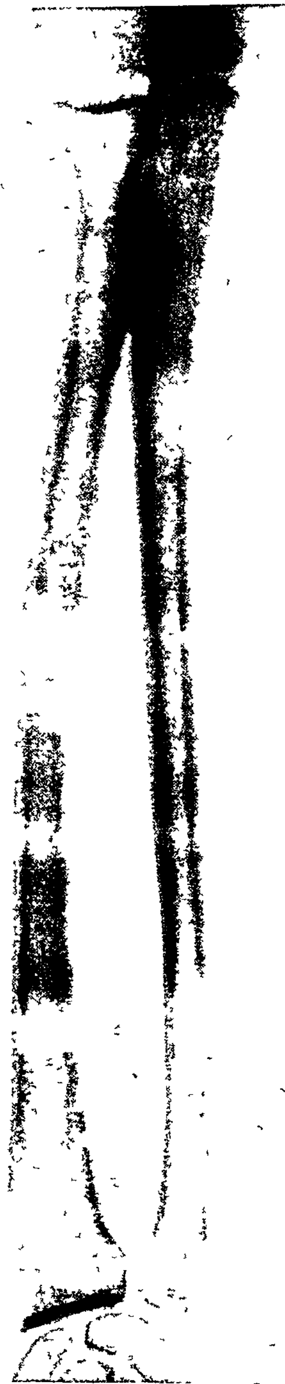


FIG 553

was first seen in London with the fractures still un-united the forearm measured less than half the normal length The wrist joint was in astonishingly close proximity to the elbow There had been infection, and the bone ends were spindled; the disability was very serious and a leather gauntlet was worn Nevertheless function of the fingers and hand was excellent and, despite the many failures, another attempt to secure union was obviously worth while (Fig 552) The un-united fracture of the ulna was grafted with an onlay tibial graft secured by screws, and with iliac bone chips Several of the earlier operations had failed by reason of infection, and penicillin was given prophylactically for three weeks Nevertheless there was minor infection, and after the fracture had united the screws were removed At a second operation another tibial graft was fixed to the radius, it was impaled into the distal fragment and fixed to the proximal fragment with one screw (Fig 552) Although the limb is still less than half the length of the other forearm, which is shown in Figure 553, the function is excellent Power of the fingers and hand is normal, and there is almost normal movement of the wrist, radio-ulnar and elbow joints

CANCELLOUS CHIP GRAFTING FOR FAILED ARTHRODESIS OF THE ANKLE JOINT (FIGS. 554-557)



FIG 554



FIG 555



FIG 556





FIG 557

Arthrodesis of the ankle joint by the Watson-Jones technique had failed—the graft was too slender and the joint surfaces were not sufficiently broken up (Fig 554) Through a posterior exposure a wedge was cut out of the back of the joint and was packed with cancellous bone chips transplanted from the ilium (Fig 555) Early union was rapid (Fig 556) and after several months consolidation was sound, as shown in Fig 557 *(Operation by Mr Lawson Dick, now of Edinburgh)*

COLLES' FRACTURE UN-UNITED AFTER EIGHT YEARS—SUCCESSFUL RESULT FROM CANCELLOUS CHIP GRAFTING (FIGS. 558-559)



FIG. 558



FIG. 559

This compound Colles' fracture had been un-united for eight years (Fig 558) In such cases the distal radial fragment is so small that it is difficult to secure sound fixation Sometimes it may be necessary to sacrifice wrist movement and perform radio-carpal fusion with a cortical bone graft onlaid from the radius to the base of the third metacarpal, but obviously it is better to preserve the wrist joint if possible In this case the lower end of the ulna was first excised to produce a radio-ulnar arthroplasty The proximal fragment of the radius was then sharpened and impaled into a small drill hole in the small distal fragment, the junction was packed tightly with cancellous bone cut from the ilium After immobilisation in an above-elbow cast for nearly four months, sound union was gained The function of the limb is now excellent (Fig 559)

BONE INFECTION—ACTUAL OR POTENTIAL—THE CERTAIN INDICATION FOR CANCELLOUS CHIP GRAFTING WITHOUT COMPACT BONE OR SCREW FIXATION (FIGS. 560-564)



Fig. 560



Fig. 561



Fig. 562

A shell-wound caused an un-united gap fracture of the ulna (Fig 560) After early infection the wound healed, and twelve months later an onlay cortical graft was applied with four vitallium screws (Fig 561) The wound of operation healed by first intention, but three months later there was radiographic evidence of bone resorption round the second lowest screw—and this was evidence in itself of grumbling infection (Fig. 561—see also p 216) A few weeks later, while the limb was still in plaster, the patient reported that there had been a crack and he complained of pain—the graft had fractured near its upper end (Fig 562) At the second operation *it was found that the proximal* and distal parts of the onlay graft had fused to the host bone but the central part was avascular, necrotic and dead—and it was surrounded by a teaspoonful of pus The dead part of the graft was



Fig 563



Fig 564

excised and the four screws were removed Pus was swabbed out and the gap was filled with cancellous chips cut from the ilium Despite low-grade infection the bone regenerated without sequestration (Fig 563) and sound union of the fracture was gained (Fig 564) When there is actual or potential infection only cancellous grafts should be used Compact bone, metal screws or wire, serve as sequestra and cause persistent infection, but cancellous bone can regenerate despite low-grade infection

CANCELLOUS BONE GRAFTING FOR FUSION OF AN ANKLE JOINT DESTROYED BY INFECTION (FIGS. 565-566)

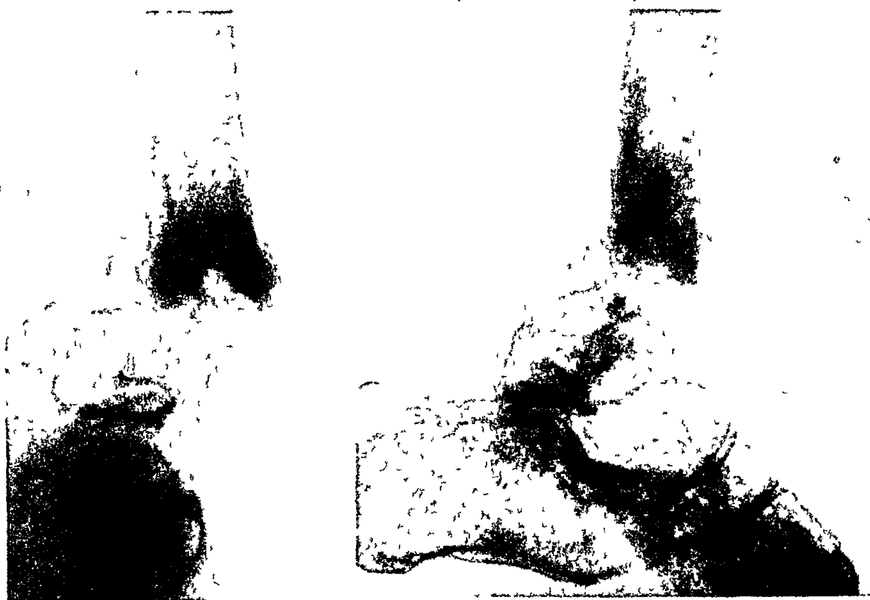


FIG. 565

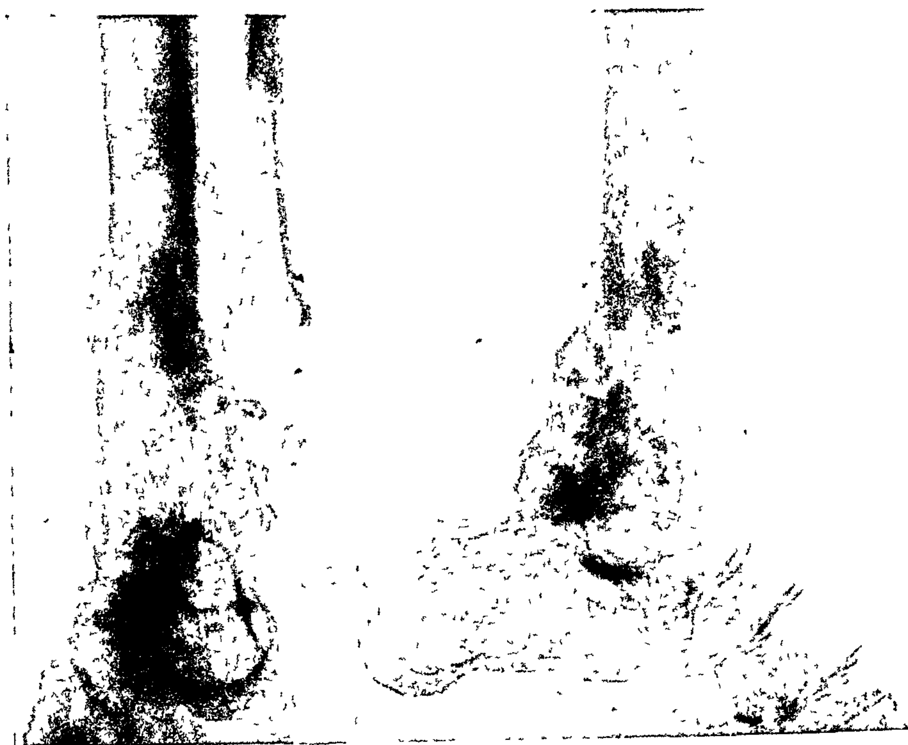


FIG 566

A woman from Israel sustained an infected wound of the ankle joint from bomb injury, much of the tibia had been excised (Fig 565) There was still infection with sinuses and adherent scars encircling the limb Six months after sequestrectomy and plastic repair the ankle was fused by operation No compact bone was used because there was still danger of potential infection With cancellous bone chips implanted between the tibia and the talus, and two iliac grafts onlaid without screw fixation, it proved possible to fuse the joint soundly and also to lengthen the limb by more than one inch. Let it not be said that there is difficulty in bridging gaps—or that bones must necessarily be impacted into each other if they are to unite (*Operations by Sir Archibald McIndoe, Mr H Osmond Clarke and R W -J*)

CANCELLOUS CHIP GRAFTING WITH INTRAMEDULLARY NAIL FIXATION SUCCEEDING AFTER FOURTEEN PREVIOUS OPERATIONS HAD FAILED FOR A FRACTURE OF THE SHAFT OF THE HUMERUS (FIGS. 567-568)

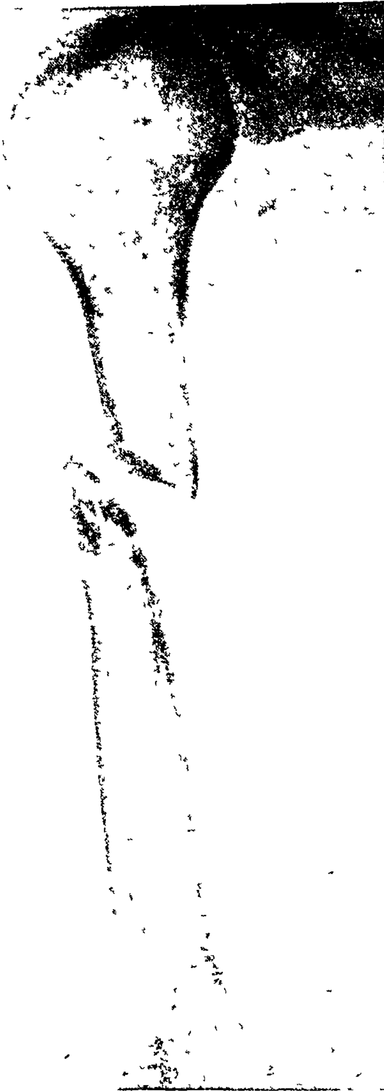


FIG 567



FIG 568

This patient had sustained a fracture of the shaft of the humerus ten years before, fourteen operations had been performed without success—the fracture was still un-united (Fig 567) The bone ends were freshened, iliac bone chips were implanted, and intramedullary nail fixation was used to supplement the protection of a plaster spica for three and a half months This technique of cancellous bone grafting with intramedullary nail fixation will be used increasingly for un-united fractures A whole-thickness compact graft with screw fixation has less mechanical efficiency than an intramedullary nail, and it has less osteogenetic activity than cancellous bone.

CANCELLOUS BONE GRAFTING WITH INTRAMEDULLARY NAIL FIXATION (FIGS. 569-570)

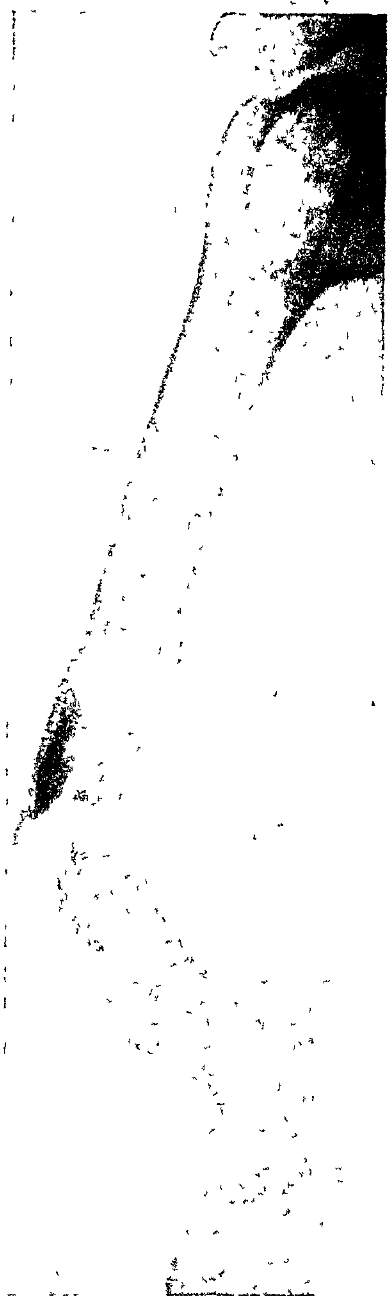


FIG. 569





FIG 570

A woman aged thirty-six years came from the Middle-East with an un-united fracture of the humerus She had lost count of the number of operations that had been performed over a period of thirteen years, she thought that there had probably been sixteen, with transplantation from both tibiæ, one femur and both sides of the pelvis All had failed because immobilisation had been inadequate (Fig 569) After simple freshening of the fractured surfaces and intramedullary nailing with impaction of cancellous iliac bone between the host fragments, combined with a plaster spica for four months, the fracture united—and there was never any doubt about it The humerus may be a cancellous bone not ideally suited to medullary nailing, and the nail used in this case may not have filled the medulla, but this degree of intramedullary fixation together with a plaster spica gave sound fixation of the bone Freshened bone will always unite if it is held long enough in rigid apposition

CANCELLOUS CHIP GRAFTING WITH MEDULLARY NAILING FOR UN-UNITED SUBTROCHANTERIC FRACTURE (FIGS. 571-572)



FIG 571

FIG 572

This subtrochanteric fracture had been treated in Switzerland with a plate and four short screws, without external fixation —and of course it failed to unite (Fig 571). What else was to have been expected from such inadequate fixation? The bone ends were freshened, an intramedullary nail was inserted, and iliac chips were impacted firmly between the bone ends No external fixation was used, but weight-bearing was deferred for more than three months The fracture united soundly (Fig 572) This patient is a very distinguished scientist, in the decomposing blood of his dressing he was able to distinguish the smell of butyric acid on the third day from that of caproic acid on the sixth day In later months he assures me that the nail served as an "alcoholometer" —within five minutes of taking wine he had aching discomfort in the thigh! The nail has now been removed and he can take sherry in comfort

PART II

FATIGUE OR STRESS FRACTURES
BIRTH FRACTURES AND
PATHOLOGICAL FRACTURES

CHAPTER XV

FATIGUE OR STRESS FRACTURES

If an aeroplane crashes in an uncontrolled power-dive the human body is disintegrated into small pieces, and the skeleton is shattered into fragments no larger than the head of a femur or the phalanx of a thumb. Between such an extreme limit of violence, and the other limit of trivial injury from ordinary weight-bearing activity, there may be any degree of bone damage from gross comminution to simple fragmentation, from fractures with overriding to fractures without displacement, from crack fractures that are recognisable at the time of injury to minute fissures disclosed in radiographs only after several weeks. Examples of fractures suspected on clinical grounds, but not at first obvious on radiographic examination, are shown on page 153 (fracture of the carpal scaphoid), page 146 (fracture of the second metatarsal), and elsewhere in these volumes.

Furthermore, it should be recognised that fractures are not necessarily "spontaneous" in origin when there is no external violence. Simple functional activity, such as walking over cobblestones or crossing a ploughed field, may crack a bone by muscle contraction alone. Fractures near the base of the fifth metatarsal, first described by Robert Jones, are typical; the injury is caused by contraction of the peroneus brevis against the resistance of an inverting foot. The patient illustrated in Figure 573 did not fall or stumble; there was no injury in the ordinary sense; but the sudden pain of which she complained arose from a true fracture produced by a single stress. Playing the piano or smoothing the sheets of a bed are not to be regarded as "injury," but either may fracture the terminal phalanx of a finger by the traction of the extensor tendon. Similarly fractures of spinous processes in the upper dorsal spine occur during the simple act of shovelling—and indeed this injury is known as the clay-shoveller's fracture.

Concealed crack fractures—Much of the confusion that abounds in the literature, including for example the use of such terms as "pseudo-fracture," and the attribution of radiographic findings in these injuries to "non-suppurative osteomyelitis,"[1] or even the diagnosis of sarcoma of bone in one march fracture,[2] has arisen from the unwillingness of surgeons to admit these two facts: 1) that ordinary functional activity may constitute an injury sufficient to cause acute fracture; 2) that crack fractures may not be disclosed in radiographs until the fissure is widened by reactionary osteoporosis or until bone is laid down beneath the œdematous and slightly raised periosteum. March fracture of the second metatarsal bone may be

[1] Weaver, J. B., and Francisco, C. B. "Pseudo Fractures: A Manifestation of Non-suppurative Osteomyelitis." *J. Bone Joint Surg.*, 1940, 22, 610.
[2] Dodd, H. "Pied Force or March Foot." *Brit. J. Surg.*, 1933, 21, 131.

of this type. The injury is often sustained by a soldier after long marching. He is tired: muscle control is imperfect; and from a single specific injury such as an awkward or uncontrolled step he cracks the bone by the impact of body-weight. Radiographs taken at once may show no proof of the fracture, but if the films are re-examined after six or eight weeks and placed alongside later films which display the exact site of injury it is often possible, perhaps with the aid of a magnifying lens, to identify the fissure that was present even in the first films.

Fatigue or stress fractures—In addition to these fractures from minor injuries—cracks so fine that at first they are concealed—bone may also be



Fig. 573

Jones's fracture near the base of the fifth metatarsal. There was no external violence; the patient was walking over a rough field and suddenly complained of pain—but it was a true fracture from a single injury sustained in the course of ordinary weight-bearing activity.

fractured by the summation of even lesser injuries. A fatigue fracture is an apparently spontaneous fracture of normal bone resulting from the summation of stresses, any one of which by itself would have been harmless. There is a precise analogy in the fatigue of metals. A metal rod may be strong enough to resist a stress of certain magnitude, but not strong enough to resist repeated applications of the same stress: the summation causes fatigue fracture of the metal. Detlefsen [1] described a fracture of the lower shaft of the fibula in a woman whose work demanded firm pressure of one foot on the vibrating pedal of a machine for eight hours each day. Such fractures may also develop in the lower shaft of the fibula in middle-aged women whose domestic life demands long standing and walking; or in the upper shaft of the fibula in adolescents and young recruits who engage in gymnastics and jump repeatedly from the crouched position.[2,3] In the records of German Army Hospitals for the year 1935-36 [4] there were

[1] Detlefsen, M. *Münch med Wschr*, 1941, 88, 303
[2] Burrows, H. Jackson. *Brit J Surg*, 1940, 28, 82
[3] Burrows, H. Jackson. *J Bone Joint Surg.*, 1948, 30-B, 266
[4] Asal, W. *Arch klin Chir*, 1936, 186, 511

nearly six hundred fractures ascribed to overloading injuries, including metatarsals, 488, tibia, 70, fibula, 12, shaft of femur, 7, neck of femur, 6; os calcis, 4, pelvis, 3 Fatigue fractures have also been described in the clavicle, first rib and ulna [1]

March fracture of the metatarsal—A march fracture of the metatarsal may thus arise from a single stumbling movement; but more generally it is a true fatigue fracture occurring in soldiers during long route marches The fracture is usually in the distal part of the second metatarsal near the neck Symptoms may develop so gradually that the patient cannot believe that he has suffered injury and sometimes it is obvious that the fracture is many weeks old when advice is first sought It may even be the "lump" of which the patient complains—a lump consisting of ensheathing callus.

Congenital shortening of the first metatarsal is a predisposing cause because the first metatarso-phalangeal joint lies at a proximal level and may even be in line with the necks of the second and third metatarsals In a normal foot the whole row of metatarso-phalangeal joints is to be regarded as a single joint at which dorsiflexion occurs with each step. In congenital shortening of the first metatarsal (metatarsus atavicus) the axis of movement between the first and fifth metatarso-phalangeal joints crosses the necks of the second and third metatarsals, and as the patient steps forwards there is an angulatory stress on these bones with 150 pounds of body-weight superimposed (Fig 574) No wonder they sometimes fracture! Other predisposing architectural weaknesses of the forefoot include hypermobility of the first metatarsal, adduction of the first metatarsal, and transverse flat foot.[2] Not uncommonly, fatigue fractures of the second metatarsal occur several weeks after the operative correction of hallux valgus deformity With hallux valgus there is already splaying of the metatarsals and transverse flat foot, and the muscle control is impaired still more by the immobility that follows operation Early exercises for the transverse arch with sustained active flexion of all toes is an important measure in after-treatment, and the exercises should be practised regularly before weight-bearing is resumed.

The first sign of march fracture is tenderness over the neck of the second metatarsal bone with slight œdema and swelling of the forefoot. The thickening then becomes harder, and after several weeks a lump can be felt at the site of callus formation In the early stages reliance must be placed on the clinical signs If a patient complains of pain in this situation after strenuous exercise, and there is localised tenderness and swelling, he

* **Spontaneous dislocation of the toes in hallux valgus**—One other complication of hallux valgus may be mentioned The metatarsal flat foot that always accompanies hallux valgus causes clawing of the toes with hyperextension at the metatarso-phalangeal joints Sometimes this hyperextension contracture progresses to such a degree that the proximal phalanx of the second toe is pulled off the back of the metatarsal head by the tight extensor tendons The metatarso-phalangeal joint is dislocated and prominence of the metatarsal head in the sole causes severe pain on attempted weight-bearing The head of the metatarsal should be removed (although some surgeons prefer to remove the base of the dislocated phalanx, as in Kellar's operation on the great toe) Usually it is the second toe that is dislocated spontaneously, but sometimes the third toe is also subluxated or dislocated It is salutory to reflect that one surgeon who operated on a bunion without recognising that there was also spontaneous dislocation of the second toe was threatened with medico-legal proceedings because the patient knew of no injury and blamed him for the dislocation

[1] Kitchin, I D *J Bone Joint S g*, 1948 **30-B**, 622
[2] Bruce, J "Anomalies of Forefoot in Relation to Metatarsal Disturbance" *Edin med J*, 1937, **44**, 530



FIG 574

March fracture of the second metatarsal with considerable callus formation when first seen—the fracture was several weeks old Note congenital shortening of the first metatarsal which predisposed to the fracture (see text).

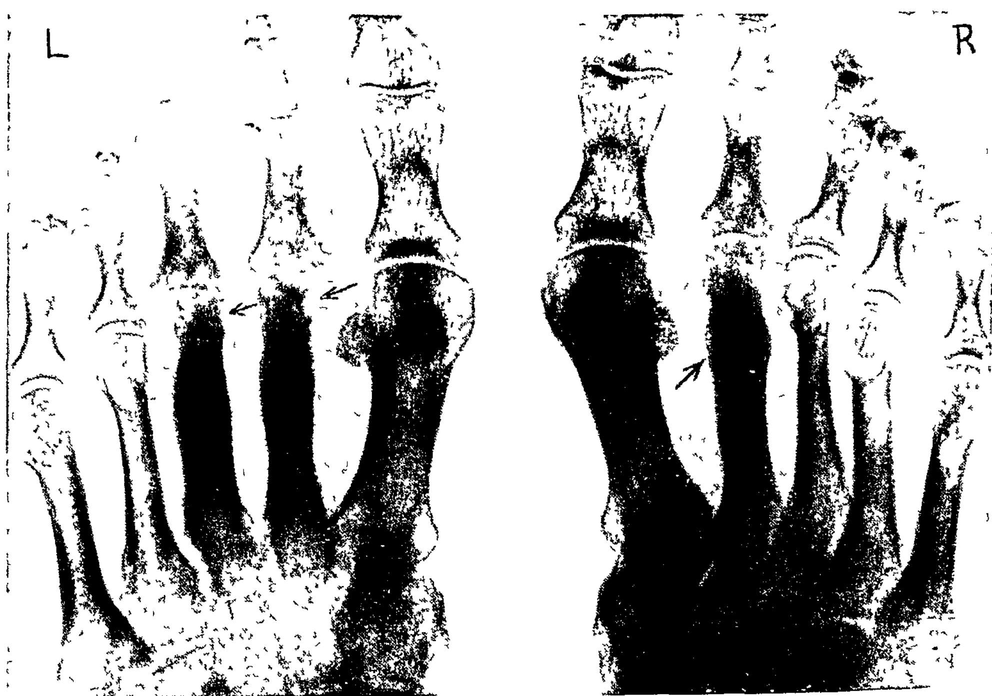


FIG 575

Panner's disease of the metatarsals In the right foot thickening of the second metatarsal shaft simulates an old march fracture, but in the left foot the changes in the second and third metatarsal heads make the diagnosis clear

has indeed sustained a march fracture of the metatarsal no matter what the radiologist may report. A walking-plaster should be applied until union is firm. After five or six weeks, simple strapping will suffice. Exercises should be taught—especially active flexion of the toes by which to redevelop the muscles of the transverse arch. If symptoms return and there is a congenitally short first metatarsal bone with recurrent fracture, it is justifiable to remove the second and perhaps the third metatarsal heads in order to realign the metatarso-phalangeal joints.

One case has been recorded of amputation of the foot for a march fracture that was mistaken for a sarcoma.[1] If sarcoma of the metatarsals was not so rare as to be almost unknown, periosteal new bone formation occurring without history of injury might be confusing. But since nearly five hundred march fractures of the second metatarsal were recorded in one year alone in one country, and sarcoma of this bone has not yet been reported in thousands of years in any country, we should be cautious in considering amputation. A more difficult differential diagnosis is from Panner's disease of the second metatarsal—an allied disorder in which there is periosteal thickening of the shaft of the second and sometimes the third metatarsals. In this condition the blood supply of the distal part of the bone is impaired, and there is usually avascular necrosis of the metatarsal head with rigidity of the joint (Fig. 575).

Fatigue fracture of the lower shaft of the fibula—This fracture occurs in active and hard-pressed women of middle age, and also in young athletes. The site of fracture in middle-aged women is usually through cancellous bone just distal to the interosseus ligament, about one and a half inches above the tip of the lateral malleolus; in young male track-racers, cross-country runners and marathon-racers, the fracture occurs through the more slender cortical bone at a slightly higher level. In each case the patient complains of discomfort and stiffness of the ankle. There is local tenderness and swelling, but radiographs may show no more than slight condensation of the bone. After a few weeks there is subperiosteal bone formation, and the line of fracture, which is always transverse, becomes more evident. It usually suffices to support the ankle with adhesive strapping and to limit weight-bearing activity, but several months may elapse before the pain is entirely relieved.

Fatigue fracture of the upper shaft of the fibula—Fatigue fracture of the upper shaft of the fibula develops in military recruits from repeated jumping; as a rule the recruit was learning to spring to the position of attack and jump from the full knees-bend position.[2] Most cases have been reported in the German literature. As long as fifty years ago Hopfengartner reported thirty-nine fractures at this high fibular level, sustained by young soldiers during their first year of service; in twelve of one series of eighteen cases, and in thirteen of another series of twenty-one, the injury was ascribed to jumping.[3] In another report, nine artillerymen sustained fatigue fracture of the upper shaft of the fibula after practising jumping from the knees-bend position for five minutes several times a day;[4] and yet another writer reported that jumping and double knee-bending had exhausted "the working

[1] Dodd, H. *Brit. J. Surg.*, 1933, 21, 131.
[2] Dreist. *Dtsch. Militärarztl. Z.*, 1909, 38, 397.
[3] Hopfengartner. *Dtsch. Militärarztl. Z.*, 1907, 36, 100.
[4] Scherf. *Zbl. Chir.*, 1933, 60, 2739.

efficiency of the bone "[1] It is interesting to note that acute fracture of the upper shaft of the fibula occurs with unexpected frequency in parachute jumpers, who usually land with the knees in flexion. In one series, which I studied, of fifty-six bone and joint injuries sustained by parachutists under training there were no less than eleven crack fractures of the upper shaft of the fibula The relationship between acute fractures from parachute landing, and fatigue fractures from military training, has been examined by Jackson Burrows[2] He points out: "As the metatarsal fracture is the typical march fracture, and the low fibular fracture is perhaps a running fracture, so the high fibular fracture appears to be typically a jump fracture"

Fatigue fracture of the tibia—There have been many case reports of fatigue fracture of the upper shaft of the tibia, usually an incomplete crack situated about three inches below the knee joint.[3] The patient complains of pain in the calf or in the upper part of the shin on the medial side. Early radiographs may show an ill-defined area of sclerosis across the shaft of the bone, and later there is a more clearly defined fracture extending incompletely through the bone with a knuckle of subperiosteal callus. The fracture may be bilateral

Multiple fatigue fractures—When the etiology is recognised it is not surprising that there have been many reports of multiple fatigue fractures, including bilateral fractures of both fibulæ at the same level,[4] or at different levels[5], low fracture of one fibula with high fracture of the other[6], fatigue fracture of the fibula with march fracture of the metatarsal[5,7], and bilateral fatigue fractures of the tibiæ[3]

Fatigue fractures of the upper limb—Fatigue fractures have been reported in the clavicle, first rib and forearm bones but with less frequency than in the bones of the weight-bearing lower limbs The fibula is not, of course, a weight-bearing bone, and fractures of this bone are essentially dependent on the stresses of muscular action Figures 576-577 show a fatigue fracture of the ulna sustained by a farm-worker, aged twenty years, who complained of pain after forking and carting manure[8] There had been no specific injury, but there was local tenderness and swelling over the middle third of the ulna accompanied after ten weeks by the typical radiographic evidence of a crack fracture with abundant callus which consolidated slowly There had never been a recognisable injury, but it was the left ulna that developed a fracture, and it was the left forearm that was the fulcrum in the physical activity of supporting the downward thrust of the fork and thereafter resisting the pull of the heavy load The limb was immobilised in plaster from the third to the thirteenth week, and the patient went back to light work four months after the first onset of symptoms

OTHER SPONTANEOUS FRACTURES

Epilepsy—The violent muscle contractions of epilepsy may cause dislocation of the shoulder, sometimes with compressed fracture of the

[1] Wachsmuth *Dtsch Militärärztl Z*, 1937, **2**, 193
[2] Burrows, H Jackson "Fatigue Fractures of the Fibula" *J Bone Joint Surg*, 1948, **30-B**, 266.
[3] Hartley, J B "Fatigue Fracture of the Tibia" *Brit J Surg*, 1942, **30**, 9
[4] Ronald, A "Fatigue Fracture" *Brit J Surg*, 1945, **33**, 90
[5] Hamilton A S, and Finklestein, H E *J Bone Joint Surg*, 1944, **26**, 146
[6] Weaver, J B, and Francisco, C B *J Bone Joint Surg*, 1940, **22**, 610
[7] Richmond, D A *Lancet* 1945, **1**, 273
[8] Kitchin, I D *J Bone Joint Surg*, 1948, **30-B**, 622





FIG 576 FIG 577

Fatigue fracture of the ulna which developed without history of injury after forking and carting manure Ten weeks after the first onset of symptoms (Fig 576) there is typically abundant callus which consolidated slowly (*By permission of Mr Kitchin and the publishers of the "Journal of Bone and Joint Surgery"*)

postero-lateral part of the humeral head so that the dislocation is recurrent In other cases the great tuberosity is avulsed by the supraspinatus In senile and bedridden patients whose bones are porotic, especially when epileptic convulsions are restrained forcibly, the long bones of the limbs may be fractured or there may be spontaneous fracture of the vertebræ.

Convulsive therapy—The convulsive therapy used by neuro-psychiatrists has been responsible for many fractures and dislocations Compression fracture of the spine is perhaps the most common, it is said to have occurred in at least 40 per cent of patients so treated[1] More than thirty fractures

[1] Palmer, H A "Vertebral Fractures in Convulsive Therapy" *Lancet*, 1939, 2, 181

of the femoral neck have been reported, many having been nailed satisfactorily [1] The violence of the muscle contraction induced artificially by cardiazol, which was once described as "the elixir of life to a hitherto doomed race," and by electrically induced convulsions, is nowadays controlled by curare, and it is unlikely that spontaneous fractures and dislocations from convulsive therapy will be seen in the future



FIG. 578

Fracture of the neck of the femur in a tabetic patient which went on to complete disorganisation and a typical Charcot's joint.

Tabes dorsalis—Degeneration of the posterior columns of the cord in tabes dorsalis causes loss of joint sensation and of pain, so that tabetic patients are not only ataxic but are also unaware of normal danger signals The unguarded gait may cause fracture of the patella or even spontaneous fracture of the tibia or the femur At early stages of the disease the fractures unite normally. It is wise to choose methods of treatment that minimise the period of bed rest, because in these patients successful ambulation depends largely on visual control which may be lost if recumbency is enforced for a long period Open reduction and internal fixation, sometimes by intramedullary nailing of shaft fractures, should therefore be performed more readily in tabetics than in other patients. In the later stages, when Charcot's joints are developing, true pathological fractures may occur By reason of the neurotrophic disturbance, which is not understood fully, the bone appears to be sclerosed and yet undergoes rapid absorption The calcaneus may appear dense and solid, but it crushes and deforms A simple fracture of the neck of the femur may result in complete disorganisation of the joint (Fig 578) Fractures are slow to unite, and it is unusually difficult—though not impossible—to gain sound fusion of Charcot's joints by surgical intervention Fortunately, there is complete painlessness, and as a rule the main problem is to restore stability, which is often best achieved by the fitting of a bucket-topped calliper splint. In any event the patient should not be kept in bed for long periods.

[1] Gissane, W, Blair, D, and Rank, B K "Fractures of the Neck of the Femur in Convulsive Therapy" *Lancet*, 1940, 1, 450.

CHAPTER XVI

BIRTH FRACTURES

Three types of fracture may occur in the newly born infant: 1) fracture or epiphyseal displacement from injury sustained during a difficult delivery, 2) multiple fractures associated with congenital fragility of bone, 3) congenital fracture of the tibia with congenital pseudarthrosis

Fractures from birth injury—The shafts of the long bones of the arm and thigh are involved more commonly than the epiphyses The injuries in order of frequency are: fracture of the shaft of the humerus, fracture of the shaft of one or both clavicles, fracture of the shaft of the femur, depressed fracture of the skull, and displacement of epiphyses of the humerus or femur The fractures are seldom of the greenstick type They are usually complete and there is often considerable displacement Large masses of callus develop and the injury may be recognised only by the lump that becomes obvious two or three weeks after birth Union is rapid, non-union is practically unknown, and even if the fragments unite with angulation alignment is restored by remodelling during the first few years of life

Fractures from congenital fragility of bone—One or more fractures of long bones present at birth may arise from osteogenesis imperfecta This will be discussed in the next chapter.

Congenital pseudarthrosis of the tibia—The pseudarthrosis of the tibia that develops at birth or soon after is characterised by curious indolence of the bones and marked angular deformity; the bowing is usually forward but is sometimes backward. It is a pathological entity of a very different type from the simple fractures of birth injury

FRACTURE OF SHAFT OF HUMERUS

Clinical features—Fractures of the middle third of the shaft of the humerus occur in breech deliveries, almost invariably during attempts to deliver the extended arms. The injury may also be produced by efforts to deliver impacted shoulders by axillary traction in vertex presentations The fracture is transverse or spiral, there is seldom overriding but often outward angulation from pull of the deltoid muscle on the proximal fragment Musculo-spiral palsy often occurs but it usually recovers within six or eight weeks There is stripping of periosteum leading to considerable callus formation and union is firm in about three weeks

Diagnosis—Dangling of an arm by the side, or failure to move the limb normally, may arise from fracture of the humerus or clavicle, Erb's palsy, or congenital syphilitic periostitis The diagnosis of fracture is established easily by clinical and radiographic examination

Treatment—The arm should be strapped to the chest over a pad of wool, thick enough to maintain moderate abduction of the limb The hand and fingers are left free. The fracture may unite with some outward angulation but this will be corrected during the first two to three years of subsequent growth Perfect remodelling of the bone is possible even if there is 40 or 50 degrees of angulation

FRACTURE OF SHAFT OF CLAVICLE

Fracture of the clavicle is usually sustained in the middle of the shaft and nearly always arises from direct digital pressure on the bone during traction on the shoulders to deliver an after-coming head. There may be displacement of the fragments similar to that occurring in adult fractures The injury often escapes notice for two or three weeks until the lump from callus formation becomes obvious Both clavicles may be fractured simultaneously. A small pad of gauze dusted with talcum powder should be placed in the axilla, between the arm and chest, and a sling be used for two weeks. No other treatment is necessary.

FRACTURE OF SHAFT OF FEMUR

Clinical features—Fracture of the upper shaft of the femur may be produced by torsion on the presenting leg in breech deliveries, but it usually occurs from efforts to deliver a breech with extended legs, either by groin traction or by bringing down the leg before delivery of the baby. The injury may also arise during extraction by Cæsarean section. The normal attitude of the new-born child is one of general and strong flexion; the hip joints are flexed to the right angle by tonic contraction of the psoas muscles The proximal fragment of a fractured shaft of the femur is therefore strongly flexed and usually lies at right angles to the distal fragment (Fig 579)

Treatment—If such a fracture is treated with the thigh extended in a Thomas' splint,[1] in a plaster spica, or in wooden spatula splints, the fragments will unite with 90 degrees of forward angulation It is true that even this degree of deformity may disappear by the time the child is six or eight years of age, but the shortening from overlap and angulation does not always disappear (Fig. 581). Credé suggested that the limb should be bandaged to the front of the infant's body with the hip joint almost fully flexed. This routine is often used, but the deformity is then over-corrected. There is backward angulation, and there may still be shortening (Fig. 580). The most rational treatment is to hold the limb at right angles to the trunk so that it lies exactly in the axis in which the proximal fragment is held by the psoas muscle. Overhead suspension of both lower limbs maintains the position and preserves full length. Several splints have been devised which achieve these objects satisfactorily [2,3,4]

The birth-fracture frame I have used is of the simplest possible type and has proved effective (Fig 582) Bandage is bound firmly over the base,

[1] Robert Jones "Treatment of Fracture of the Femur in the Newly Born" *Brit med J*, 1908, 1, 1358.
[2] Silver, D "Treatment by Suspension of Fracture of Femur in Young Children" *Ann Surg*, 1909, 49, 105
[3] Robinson, W H "Treatment of Birth Fractures of Femur" *J Bone Joint Surg*, 1938, 20, 778
[4] Ruiz Moreno, M "Obstetrical Fractures of Humerus and Femur" *Prensa med Argent*, 1938, 25, 321





Fig 579 Fig. 580

Birth fracture shaft of femur showing the usual right-angled flexion of the proximal fragment (Fig 579). Treatment by strapping the limb to the trunk over-corrects the deformity and fails to correct the shortening (Fig 580)

Fig. 581

Birth fracture of the shaft of the femur treated by strapping to the trunk, six years after injury Although the angulation is corrected perfectly by remodelling, there is still one inch of shortening Note the downward tilt of the pelvis on the injured side

and on this the infant lies ; there are two notches on the crossbar, and extension strappings from each lower limb are fastened to them The limbs must be held with sufficient tension to raise the buttocks from the bandage slings Traction on the limb may be increased by bandaging the trunk of the infant to the frame In this frame, nursing presents no problems, napkins are used in the ordinary way and there is no difficulty from pressure sores. Union is usually quite firm in three weeks and the apparatus is then discarded.

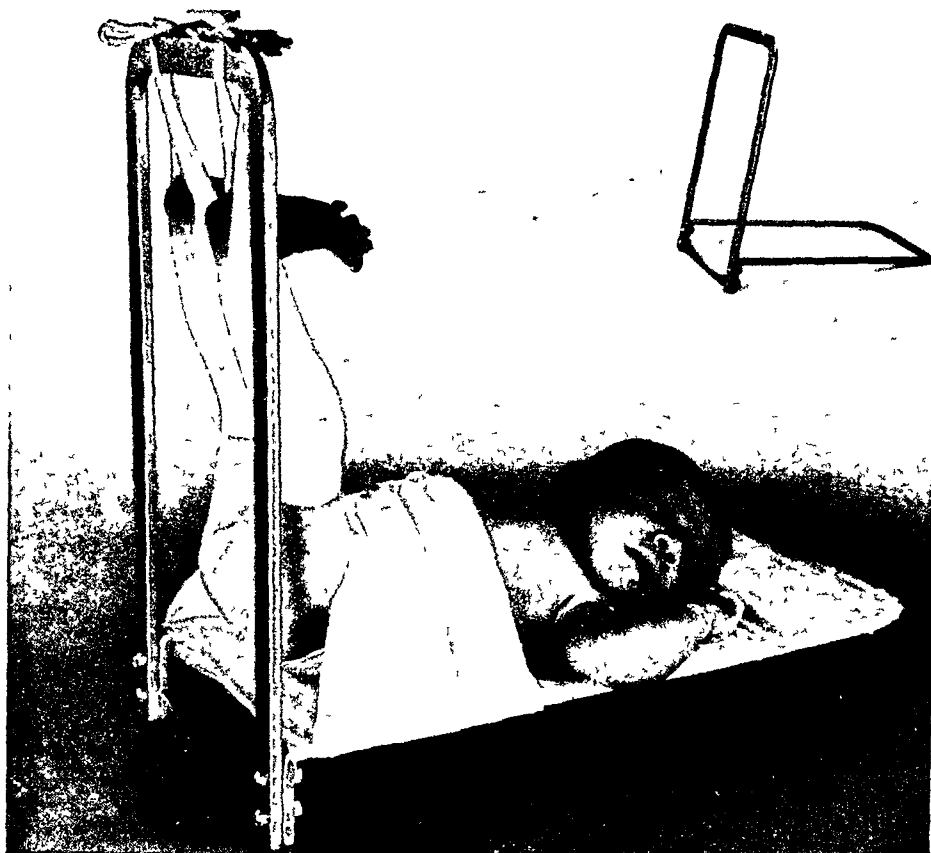


FIG. 582

Birth fracture frame for injuries of the femoral shaft The overhead suspension maintains length and keeps the limb in the direction in which the proximal fragment is held by the psoas muscle.

DEPRESSED FRACTURE OF SKULL

The pliability of the skull and the wide open sutures protect the cranial bones of the infant from fracture Even severe intracranial birth injuries accompanied by asphyxia, respiratory distress, circulatory failure, and cerebral hæmorrhage with spastic diplegia, are usually unaccompanied by fracture. If a fracture is sustained, the parietal bones are involved and the injury arises from pressure of the head on the bones of the pelvic inlet, particularly the promontory of the sacrum, in delivery through a slightly contracted pelvis The depressed area of bone usually undergoes spontaneous elevation, very occasionally, uncorrected depressions and furrows persist into adult life.

EPIPHYSEAL DISPLACEMENTS

Birth injury may cause displacement of epiphyses at the lower end of the femur, lower end of the humerus, or upper end of the humerus.[1,2]

Lower femoral epiphysis—The centre of ossification is present at birth and the radiographic diagnosis of backward displacement presents no difficulty The periosteum at the back of the lower shaft of the femur is stripped and displaced backwards with the epiphysis. If the displacement is not corrected the subperiosteal hæmatoma ossifies and a new shaft is formed behind the original one. The lower end of the original shaft is absorbed gradually The radiographic appearances become quite normal after two or three years even despite complete failure of reduction If any attempt is made to correct the displacement, care must be taken not to compress the popliteal vessels and endanger the circulation of the foot. Traction should be applied to the limb before the knee is extended. The limb should be immobilised for three weeks on a spatula splint or in a birth fracture frame (Fig 582)

Lower humeral epiphysis—The centre of ossification of the lower humeral epiphysis does not appear until the latter half of the first year. If the epiphysis is displaced backwards by birth injury, radiographic diagnosis is impossible until ten or fourteen days after birth. Ossification is then seen beneath the displaced periosteum behind and to one side of the lower shaft of the bone. The clinical diagnosis may be made shortly after birth by the signs of helplessness and immobility of the limb, pain on attempted movement, and swelling of the elbow. Traction should be applied to the limb and, while it is maintained, the elbow is flexed gently to just above the right angle and then supported in a sling. Even if the displacement remains imperfectly corrected the regenerative powers of the infant are such that an apparently normal bone is reconstructed, exactly as in injuries of the lower femoral epiphysis

CONGENITAL PSEUDARTHROSIS OF THE TIBIA

Whereas birth fractures of the femur, humerus and clavicle unite rapidly with massive callus formation, congenital pseudarthrosis of the tibia is characterised by indolence of the fragments, minimal callus formation, and non-union which often persists despite prolonged immobilisation and bone-grafting operations. One child was treated by continuous and uninterrupted immobilisation in plaster for four years without success,[3], another was operated upon fifteen times in a determined attempt to secure union ; Putti reported eleven failures in thirteen bone-grafting operations , Henderson[4] recorded seven failures in eleven cases , amputation was often performed after many years of treatment because limbs were shortened, atrophic and functionally useless

The pathology is not yet understood fully. No abnormality has been found in the calcium phosphorus or phosphatase levels of the blood ; there is no hereditary factor and the condition is seldom associated with congenital

[1] Michel L " Obstetrical Dislocation of Upper Humeral Epiphysis " *Rev Orthoped* , 1937, 24, 201
[2] Scaglietti, O 'The Obstetrical Shoulder Trauma ' *Surg Gynec Obstet* , 1938, 66, 868
[3] McFarland, B " Birth Fracture of the Tibia " *Brit J Surg* , 1940, 27, 706
[4] Henderson, M S , and Clegg, R S *Proc Mayo Clin* , 1941, 16, 769

deformities or developmental errors Patches of café-au-lait pigmentation of the skin have been reported in some cases but not in all [1] The evidence that pseudarthrosis of the tibia is not a true congenital abnormality lies in the fact that the child is often normal at birth Three types may be recognised. (1) the infant is born with a fracture of the tibia sustained before or during birth and failing to unite [2], (2) the infant is born with angular deformity of the tibia but no fracture, and pseudarthrosis develops in later months or years as the result of fracture or osteotomy [3]; (3) the infant is normal at birth but sustains a fracture of the tibia with pseudarthrosis in early childhood. It is clear, therefore, that "congenital" pseudarthrosis and "birth" fracture are misleading terms It is even open to doubt whether any congenital or constitutional abnormality exists, it seems much more likely that pseudarthrosis arises from the same local conditions that cause non-union of tibial fractures in adults. This is suggested by three features which are common to all cases of pseudarthrosis in infants the site of fracture; the angulation of the fragments; the instability of the fracture and difficulty of immobilisation The site of fracture near the junction of the middle and lower thirds of the shaft of the tibia is a level where blood supply of the bone is feeble and where fractures in adults often show marked indolence, minimal callus formation and slow union, in striking contrast with shaft fractures elsewhere. The delaying influence of a poor blood supply was discussed in earlier chapters, and it was shown that slow union from this cause often leads to non-union if there is also shearing strain and imperfect immobilisation. One important source of shearing strain which has gained full recognition only recently is weight-bearing on an angulated bone.[4] It is obvious that this applies also to tibial fractures in infants because a marked degree of angulation is almost invariable in congenital pseudarthrosis, and early weight-bearing has long been encouraged in the hope of stimulating union. Moreover, the factor of imperfect immobilisation, known to be so important in adult fractures, is still more evident in the fractures of infants. Even when there is no fracture, it is most difficult to immobilise the elastic bones and joints of infants. Every orthopædic surgeon is aware of the incredible facility with which a baby can wriggle his limb out of a plaster applied from toes to groin with right-angled flexion of the ankle and knee joints How much more readily can a child wriggle his limb when there is also a fracture, especially when it is a fracture with complete lack of natural stability owing to the conical shape of the pointed fragments and the intervening gap. In the infant, no matter what care is used, it is impossible to immobilise a fracture of the tibia completely by the external fixation of splints or plaster Even the internal fixation of a bone graft is inadequate if a single autogenous graft is used because the bone is so slender and fixation so difficult. If it is true that the union of fractures of the shaft of the tibia in infants is dominated by the same influence of blood supply as the union of fractures in adults, and that the same complete and prolonged immobilisation is needed, the source of congenital pseudarthrosis is explained. This would be confirmed if a new method of grafting was devised and it was found that complete internal fixation and complete protection from angulatory

[1] Moore, B H "Orthopædic Relationship of Neurofibromatosis" *J Bone Joint Surg*, 1941, 23, 109
[2] Camurati, M "Le pseudartrosi congenite della tibia" *Chir Organi Mov*, 1930, 15, 1
[3] Hallock, H. "Pseudarthrosis following Osteotomy for the Correction of Congenital Deformity" *J. Bone Joint Surg*, 1938, 20, 648
[4] Watson-Jones, R, and Coltart, W D "Slow Union of Fractures" *Brit J Surg*, 1943, 30, 260

strain was always successful in securing union. Recent experience suggests that this may now have been accomplished, and it supports the view that failures in the past occurred not from obscure pathological changes in the bone but from imperfect operative technique, and reliance on slender grafts inadequately fixed to angulating bones.[1] [2]

The first point in the technique is that an inlay graft can seldom hope to succeed because it is necessarily more slender than the very slender host bone, moreover, the preparation of a bed for an inlay graft involves excision of the conical ends of the fragments and serious increase in the gap between them. An onlay graft must be used so that all host bone is preserved (Fig 583) In the second place, the fixation of a single graft to atrophic attenuated fragments presents almost insuperable difficulty, and it is doubtful whether such grafts were ever completely successful in controlling angulatory strain. Double onlay grafts must be used, one on each side of the pseudarthrosis, fixed securely to each other with the host bone clamped between them as if in a vice Sufficient bone for dual grafts cannot always be secured from the infant, and maternal grafts or bone from a bank are usually necessary. Finally the fixation of grafts, prevention of recurrent angulation, and control of strain at the site of pseudarthrosis must no longer depend on the devices formerly used—fragments of wire and bits of catgut that any carpenter would laugh to scorn. Screws which completely fix the grafts and transfix the host bone must be used. Stainless steel, vitallium and other alloys have made it possible to use screws without fear of reaction or of loosening in the bone This development in surgical equipment has made the modern technique of onlay grafting possible and provided the key to the problem of congenital pseudarthrosis of the tibia

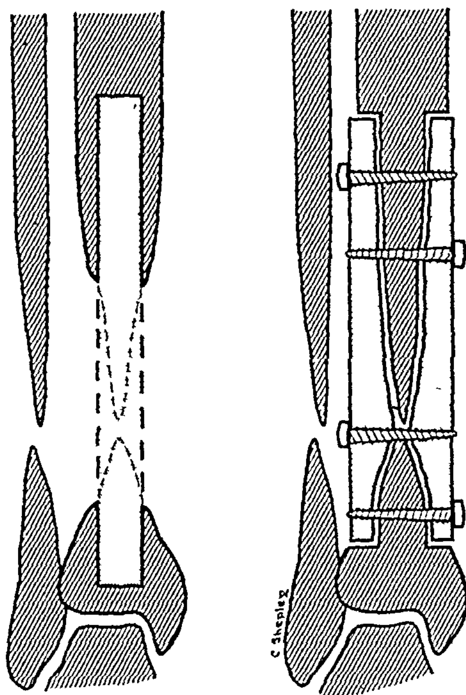


FIG 583

In congenital pseudarthrosis of the tibia an inlay graft is difficult to fix, and it seriously increases the gap between the fragments. These difficulties are met by double onlay grafts screwed to each other

Before the introduction of vitallium screw fixation for double onlay grafts, Macfarland [3] recorded successful results from a by-pass technique of bone grafting (Fig. 587) Without attempting to correct the deformity, a massive graft was wedged on the concave side of the angulated bone at a distance from the pseudarthrosis which was bridged by the graft. He believed that one reason for his success might be the lack of direct contact between graft

[1] Boyd, H B "Congenital Pseudarthrosis and Treatment by Dual Bone Grafts" *J Bone Joint Surg* 1941, 23, 497

[2] Henderson in reporting four successes in eleven operations, records that even in the successful cases "the grafts broke and the patients seemed doomed to go on with non-union, but slowly, under supportive measures and the use of braces over a long period, union developed" In the other seven cases failure was complete Could more honest acknowledgment be made of the failure of operative technique in former years? "Pseudarthrosis of the Tibia" *Proc Mayo Clin*, 1941, 16, 769

[3] McFarland, B "Birth Fracture of the Tibia" *Brit J Surg*, 1940, 27, 706 (This article also records success from massive double onlay grafting, but vitallium screw fixation was not then employed)



Fig. 584

Congenital pseudarthrosis of the tibia.

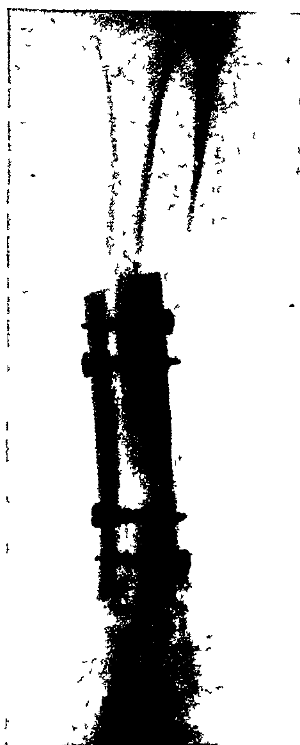


Fig. 585



Fig. 586

An attempt to bridge the pseudarthrosis by transposing the fibula failed Double onlay maternal grafts were used (Fig. 585) After immobilisation in plaster for nine months union was soundly consolidated (Fig. 586)

and pseudarthrosis where it appeared that osteolysis was almost malignant in its capacity to absorb all bone within reach. The true importance of his technique was the sound fixation of a stout graft to a broad base of bone well above and below the site of pseudarthrosis in such a position that the inhibitory effect of angulatory strain was excluded by short-circuiting

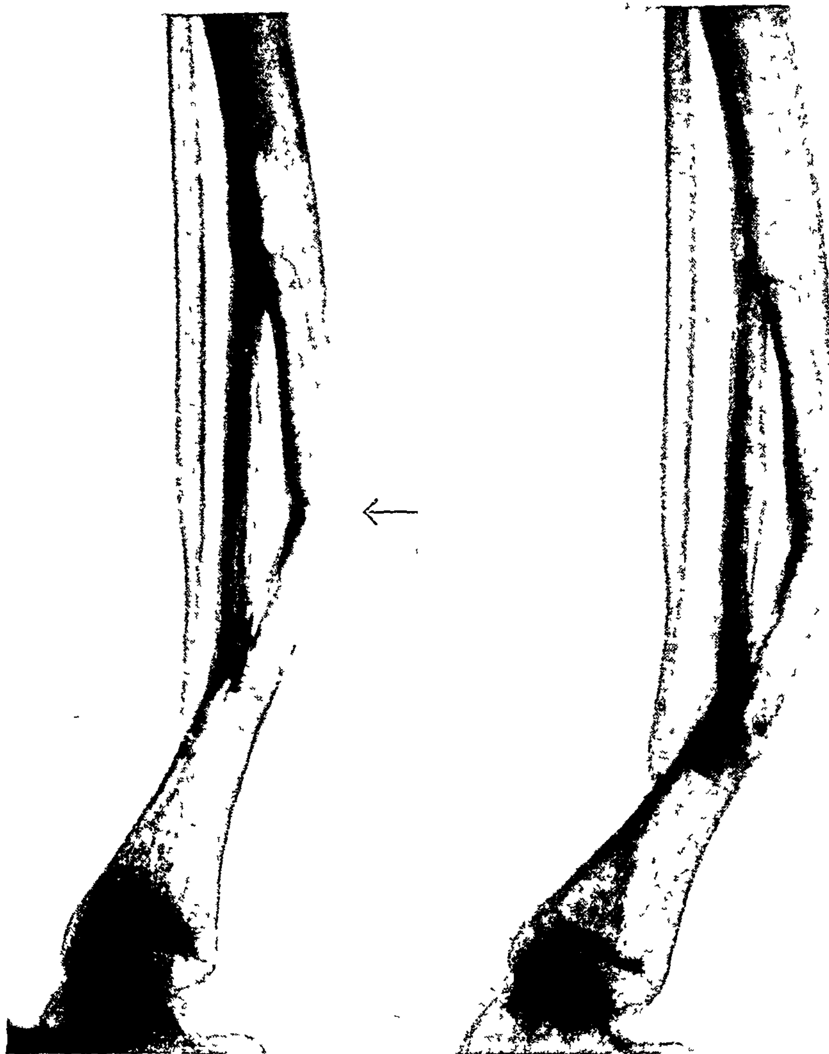


Fig 587 Fig 588

A congenital pseudarthrosis of the middle third of the tibia was treated by a bridge graft and it united (Fig 587), but at the lower end of the graft where there was still angulatory strain due to the forward bow of the tibia a second pseudarthrosis developed (Fig 588), presumably at the site of a fine crack sustained during the grafting operation.

the angulation This is confirmed by an interesting case in which I performed Macfarland's operation The pseudarthrosis was bridged by a stout graft so that the line of weight-bearing passed through the graft and no longer through the angulated bone. The pseudarthrosis united promptly; but the degree of angulation of the tibia was such that weight-bearing still passed in front of the ankle joint; there was still a forward bow of the tibia at the site of impaction of the lower end of the graft, and at this level a second pseudarthrosis developed (Figs 587-588). It is presumed that a slight

crack was sustained at the time of the grafting operation and was slow in healing, so that after several months, with the renewal of weight-bearing and angulatory strain, the crack widened to an obvious fracture and developed all the characteristics of " congenital " pseudarthrosis When a further operation was performed, alignment was corrected at the second pseudarthrosis so that weight was transmitted accurately through the tibia from knee to ankle, and fixation was maintained by onlay grafts screwed to the host bone. This operation was completely successful, because accurate restoration of alignment had excluded angulatory stresses. *The secret of the treatment of congenital pseudarthrosis of the tibia is to gain sound fixation with the host fragments in accurate alignment*

CHAPTER XVII

PATHOLOGICAL FRACTURES

Traumatic surgery cannot be practised in isolation from general orthopædic surgery—this is most clearly shown in the study of pathological fractures A fracture may be the earliest manifestation of genetic disturbance, hormonal imbalance or vitamin deficiency, of primary or secondary tumour of bone, or of lipoid granulomatosis, leukæmia or other disease of the marrow constituents Changes in the structure of bone which at first seem to be localised are often found to be part of a general disease for example, expansion and porosis of the shaft of a fractured bone may be the first sign of hyperparathyroidism, fractures of ribs from trivial injury may be evidence of Cushing's syndrome or cœliac disease; compression of a single sclerosed vertebra may lead to the diagnosis of Paget's disease, carcinoma of the prostate or lymphadenoma

Surgeons who treat fractures must be alert to the many possible implications of bone injury, and particularly to the recognition of dystrophies of bone with predisposition to fracture, including: developmental errors of growth; nutritional and vitamin deficiencies, disorders of hormonal imbalance, disuse and senile atrophy; cysts, fibrous dysplasia, and Paget's disease, primary and secondary tumours, diseases of the marrow constituents, infections and parasitic diseases; and neurotrophic dystrophies of bone

DEVELOPMENT AND GROWTH OF NORMAL BONE

In understanding diseases of bone in their relation to pathological fracture we should first recall the development, growth and maintenance of normal bone Bone is a living tissue depending for its metabolism on living cells known as osteocytes, which lie in oval-shaped lacunæ, with long branches passing through minute canaliculi to join similar branches from other osteocytes. There is an intracellular matrix of collagenous fibrillar structure with a cementing substance of bone-salt including calcium, phosphate and carbonate ions In addition to these permanent elements there are transient elements, observed during active phases of bone formation and bone destruction, namely osteoblasts and osteoclasts.

The cells of bone—*Osteoblasts* are not cells of specific origin with an inherent destiny to form bone, they are derived from primitive undifferentiated mesenchymal cells of the reticulum of marrow, endosteum, Haversian systems and subperiosteal layers Under the stimulus of functional activity, and the influence of hormones and vitamins, they become differentiated as bone-forming cells and, by virtue of function rather than origin, are known as osteoblasts Osteoblasts grow and multiply in a continuous layer upon the surfaces of primitive cartilage and mesenchymal condensations in the embryo, and upon the trabecular surfaces of old bone in the adult They lay down osteoid, which is the uncalcified organic basis of immature new bone *Osteoclasts* have the function of removing

bone, and they too are derived by differentiation from primitive mesenchyme to become multinuclear giant cells with from twelve to twenty nuclei, and sometimes with as many as one hundred. They lie singly, or sometimes in small groups, in shallow hollows known as Howship's lacunæ eroded from the surfaces of bone trabeculæ. *Osteocytes*, like other living cells, have a limited span of life, but unlike other cells they cannot divide and multiply by mitosis Having no hope of posterity they must be removed, together with all surrounding bone, before they can be replaced by young osteocytes in new bone Continued osteoclastic resorption and osteoblastic replacement is therefore an essential feature of the survival of bone and its osteocytes

Three types of ossification—Bone can be laid down only upon a preliminary scaffold which may consist of cartilage, membranous mesenchyme, or old bone; and correspondingly there are three types of ossification—1) endochondral ossification; 2) membrane ossification; 3) endosteal or appositional ossification *Endochondral ossification* may be observed in the shafts of embryonic long bones which are preformed in cartilage. Bone is deposited as an encircling cuff beneath the perichondrium, the enclosed cartilage cells swell, the intercellular substance calcifies, and the cartilage cells are then removed and replaced by young bone. Likewise, throughout skeletal development and until late adolescence, there is endochondral ossification in epiphyseal plates with a similar sequence of multiplication of cartilage cells, swelling of cells, calcification of matrix, and replacement by bone—represented by the four layers of an epiphysis the layer of resting cartilage cells near the end of the bone; the zone of proliferating cartilage cells lying in orderly columns, the layer of more mature cells which are swollen and separated only by a slender intercellular matrix, and the zone in which there is calcification of matrix in preparation for the removal of cartilage cells with replacement by irregular trabeculæ of immature bone The weakest layer of the plate is the third, where strength depends entirely on slender walls of uncalcified matrix, and it is here that epiphyseal separation and displacement occur in certain hormonal disorders It should be noted that if intercellular calcification fails, as for example in rickets where insufficient calcium is absorbed from the alimentary tract, cartilage cells continue to multiply but they are not removed or transformed into bone, and the epiphyseal plates become progressively thicker *Membrane ossification*—The bones of the vault of the skull are not preformed in cartilage, from a centre of ossification in the membranous capsule of the brain slender trabeculæ of bone are laid down on mesenchymatous condensations, radiating outwards until they form a circular platelet of immature fibrillar bone. *Endosteal or appositional ossification*—As tubular bones grow in length by endochondral ossification at the epiphyses, they also grow in width by appositional ossification beneath the periosteum, new bone being deposited upon the trabeculæ of older bone Moreover, endosteal ossification continues in all parts of bone throughout life

Continued resorption and apposition of bone throughout life—Living bone is subject to continued osteoclastic resorption and osteoblastic replacement Modelling resorption, which is characteristic of early skeletal growth, was recognised long ago by John Hunter. He pointed out that the shaft of a long bone flared out to a wider epiphyseal disc; that as bone grew in length, the site of the wide metaphysis of earlier years corresponded

in level to the narrow shaft of later years, and that this could be achieved only by the removal of bone already laid down. Similarly, the excess of callus surrounding a recently united fracture and filling the medulla is removed by modelling resorption. When a fracture unites with angulation, appositional growth on the concave side and resorption on the convex side restore the shape of the bone partly at least in the adult and almost wholly in the infant, in whom even right-angled deformity of birth fractures can be corrected. Moreover, apart from the modelling that is needed in moulding the shape of bones, there are day-to-day requirements of resorption and apposition imposed by variations in functional activity. Functional disuse causes osteoporosis, whereas increased functional activity causes apposition of bone. Until recent years it was believed that disuse rarefaction arose from "decalcification" or halisteresis, and that calcium salts were removed from the cellular matrix which remained intact, but histological study shows that this is not so. Even in simple disuse atrophy there is removal not only of calcium salts but also of the trabeculæ themselves—of the organic as well as the inorganic constituents.

Not only is bone removed when it is no longer needed—as in the modelling of growing bone, the adaptation to deformity, and the response to functional inactivity—but also when its vitality is outlived. Living cells are essential for metabolic activity, in bone as well as in other tissues, but since osteocytes are incapable of mitosis and cannot be perpetuated by succeeding generations they must be replaced after death by complete removal of the old bone, with substitution by new bone, including young and vigorous osteocytes. Failure of such replacement explains the osteoporosis of senility. Osteocytes continue to die as they have died throughout skeletal life, and the bone around them must be removed, but growth of new bone is arrested by general senile retardation of cellular proliferation, and there is increasing osteoporosis.

Disturbance of the balance between bone destruction and bone formation is seen also in the hormonal disturbances that suppress osteoblastic activity (for example in Cushing's syndrome, where there is pituitary-adrenal hyper-function and excessive secretion of adrenal gluco-corticoids, and sometimes in the cortisone treatment of rheumatoid arthritis). Such hormonal suppression of osteoblastic activity leaves unopposed the osteoclastic resorption with which it is normally balanced, and it displays clearly the continued and inexorable removal of adult bone; trabeculæ disappear and there is increasing osteoporosis, often with pathological fracture.

Growth of new bone tissue—Two essential features of the growth of new bone are: 1) the development by osteoblasts of an extracellular organic substance known as osteoid; 2) the deposition in these osteoid seams of a calcium-phosphate-carbonate salt precipitated by the action of an enzyme, alkaline phosphatase. New bone formation may fail in respect of either of these essential parts. In rickets and osteomalacia osteoid is laid down abundantly, but insufficient bone salts are absorbed from the alimentary tract and the tissue remains uncalcified. In osteoporosis there is such complete failure to lay down osteoid that no matter how freely calcium salts are available there is no ground-substance in which they can be deposited.

Conversion of immature to mature bone—Calcified osteoid formed in the development of skeletal bones, or laid down in the primary repair of fractures,

differs markedly in its structure from that of mature bone It is spongy or "woven" bone with a coarse-fibred fibrillar structure arranged in whorls around blood vessels. The mineral content is less than that of mature bone, and is distributed unevenly. The irregularly shaped osteocytes have fewer connecting branches with other bone cells. On the basis of this coarse fibrillar bone, the complex lamellated structure of mature bone is apposed, primary woven bone being removed as lamellar bone is laid down. Phylogenetic history is thus recapitulated; vertebrates in early evolution had only woven bone, whereas mammals, developing later, acquired the complex and elaborate pattern of lamellated bone. All rapidly growing bone develops first as spongy or woven bone, the trabeculæ surrounding and enclosing areas of marrow with its blood vessels. The dimensions of the enclosed marrow spaces are reduced progressively as concentric lamellæ of new bone are laid down, until finally the spaces are represented only by small canals — the Haversian canals of mature bone with their Haversian vessels.



FIG. 589
Osteitis deformans
Early stage showing deposition of osteoid on the surface of the original shaft of the tibia.

Failure of conversion of immature to mature bone is seen in many disorders—typically, for example, in Paget's osteitis deformans where during active stages of the disease the shafts of long bones are thickened by coarse-fibred bone which is not converted to lamellated bone, whereas during remissions of activity, when bone formation is slower, mature lamellated bone is developed (Figs 589-591). In osteogenesis imperfecta, where there is genetic damage to osteoblasts, coarse fibrillar bone continues to be produced long after it should have been converted to lamellated bone and, although slenderness of long bones is often seen in this disorder, the most severe prenatal cases are of the thick-boned type—the thickness consisting entirely of immature and fragile bone. In osteopetrosis or Albers-Schonberg's disease, where there is congenital deficiency of osteoclasts, the metaphyses show club-like thickening from solid masses of primitive bone in which there is so little osteoclastic activity that it is not resorbed or converted into a lamellar structure

Chemistry of bone formation—The inorganic matter of bone consists chiefly of calcium, phosphate and carbonate, and the bone salt is probably dahlite, represented by the formula $CaCO_3$, $2Ca_3(PO_4)_2$ The same three

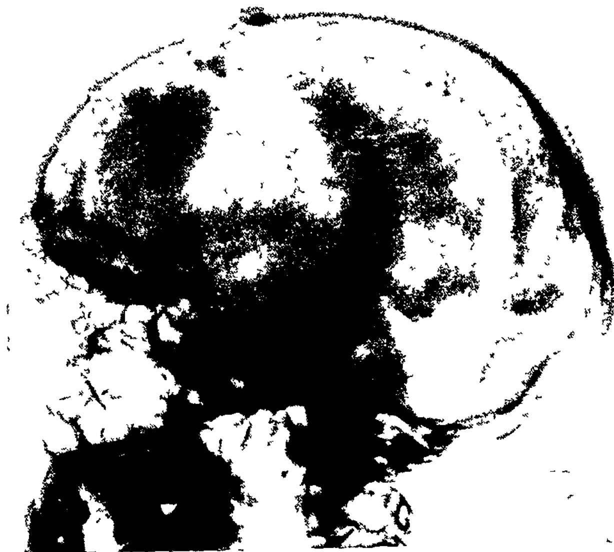


Fig 590

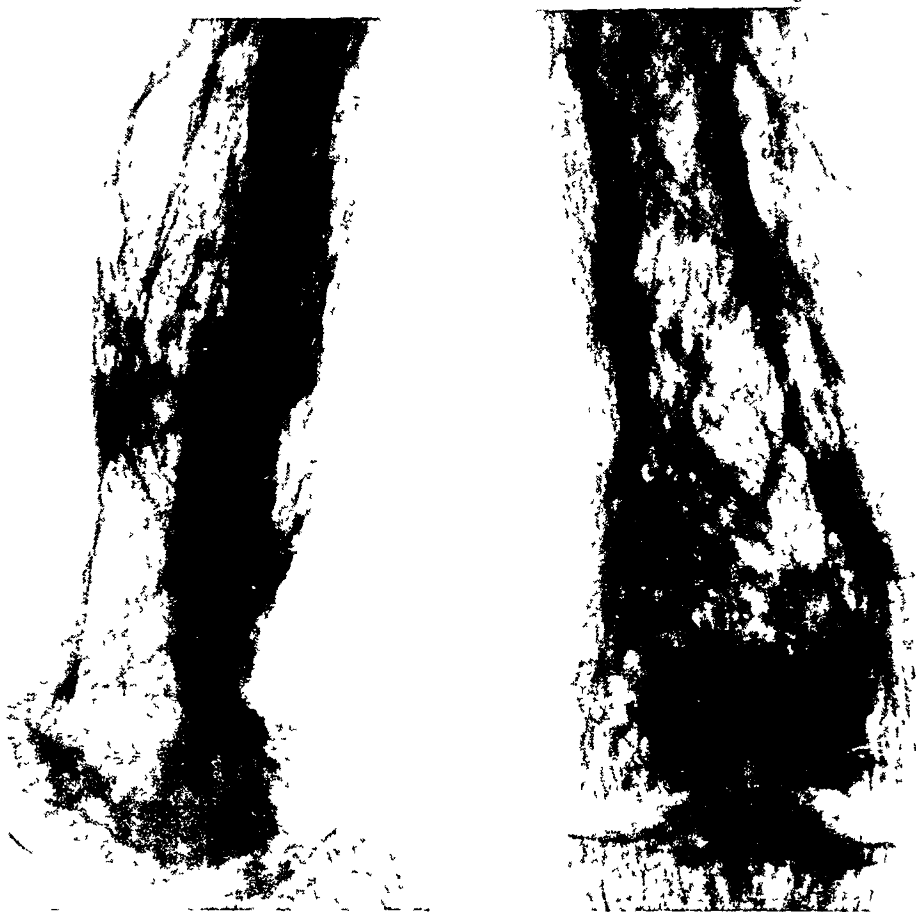


Fig. 591

Osteitis deformans

Later stage showing unbridled new bone formation in the shaft of the femur, consisting largely of osteoid and woven bone which is fragile and susceptible to fracture The skull shows porotic thickening of some bones of the calvarium from deposition of osteoid while other adjacent bones are almost normal—a change which only recently has been recognised as typical of Paget's disease

ions are present in blood serum and there is constant interchange between bones and serum.

Serum-calcium—The normal serum-calcium value is 10 mgm. per cent (plus or minus 1 mgm) At this level there is a continuous drain of calcium from the body because the renal threshold for calcium excretion is about 7 mgm per cent, and moreover there is also excretion of calcium into the intestinal tract, slight loss through the placenta in pregnancy, and considerable loss from the lactating breast. Normally, the balance of calcium is maintained by intake in the diet, but if the intake is insufficient as in starvation-malacia, or if calcium absorption from the alimentary tract is prevented by vitamin D deficiency or steatorrhœa, the blood level can be maintained only by withdrawal of calcium from the bones. Bones and teeth store more than 99 per cent. of the total calcium in the body and they represent the only supply from which blood levels can be maintained

Serum-phosphorus—Phosphorus also is found in large amounts in bones and teeth, the Ca P proportion being approximately 2·1. The serum-phosphorus level, representing inorganic or phosphate-phosphorus, is 3·2 mgm. per cent. (plus or minus 0·5 mgm., and 1 or 2 mgm. higher in growing children). But there are many organic phosphate compounds such as nucleoprotein, phospholipids and various phosphoric esters, which can be hydrolysed to liberate phosphate ions, so that a negative phosphorus balance does not necessarily deplete the bones of phosphate

Serum-alkaline-phosphatase—Osteoblasts undergoing rapid multiplication release an enzyme, alkaline phosphatase, which has the property of splitting organic phosphate compounds and producing an excess of phosphate ions so that, by exceeding the solubility product, a calcium-phosphate-carbonate salt is precipitated in crystalline form in the osteoid[1-3] Local increase of alkaline phosphatase is reflected in a general rise of the serum-phosphatase level (the normal in adults being from 3 to 5 Bodansky units; and in children, in whom bone growth is rapid and continued, up to 10 units) It should be emphasised, however, that high serum-phosphatase indicates no more than increased osteoblastic or osteoclastic activity, and that the level is high whether new formation of osteoid succeeds or fails in defence against an osteolytic process It is therefore raised in rickets and osteomalacia even when reduced calcium-intake prevents the formation of bone, and it is also raised in hyperparathyroidism where bone destruction preponderates In Paget's disease the serum-phosphatase level is higher per unit of bone disease than in any other condition, sometimes reaching a level of 60 or more units, thus indicating the tendency in this disease to unbridled bone formation On the other hand in Cushing's syndrome, and other forms of pituitary-adrenal hyperfunction where osteoblastic activity is suppressed, the serum-phosphatase level is usually low or normal, and is seldom raised

Relation of serum-calcium to serum-phosphorus—Calcium, phosphate and carbonate ions are present in crystalline form in bone and in soluble form in plasma, and there is constant interchange between the two It must therefore be accepted that for any one level of carbonate ions the

[1] Robison, R "The Possible Significance of Hexophosphoric Esters in Ossification" *Biochem J* 1923, 17, 286

[2] Robison, R, and Martland, M "The Enzyme in the Early Stages of Bone Development" *Biochem J*, 1924, 18, 1354

[3] Robison, R, and Soames, K M "The Phosphoric Esterase of Ossifying Cartilage" *Biochem J*, 1924, 18, 740.

levels of calcium and phosphate ions are dependent on a solubility product, perhaps $CaHPO_4$, which governs the relative levels of calcium and phosphate. As serum-phosphorus falls and rises, serum-calcium rises and falls, in order to keep constant the solubility product Adjustment is such that the product of serum-calcium and serum-phosphorus, expressed in milligrammes per cent, is roughly from 30 to 40 in adults (and from 40 to 55 in growing children) For example, with a normal serum-calcium of 10 mgm, and serum-phosphorus of 4 mgm, the product is 40, and in hyperparathyroidism with a serum-calcium of 20 mgm, and serum-phosphorus of 2 mgm., the product is still 40 The ratio may of course be disturbed if there is secondary renal disease

The significance of altered serum calcium and phosphorus values may be illustrated by the example of parathyroid hyperactivity causing "osteitis fibrosa cystica" This hormone has a specific effect in increasing phosphate excretion by the kidneys so that the level of serum-phosphorus tends to be lowered Equilibrium in the serum must be maintained, and is achieved by release of phosphorus from bone in the form of calcium phosphate There is therefore excess of calcium in the serum, excreted by the kidneys, so that although serum-calcium is high there is continued resorption of calcium from bones and continued excretion from the kidneys

Hormonal control of bone growth—Growth of bone is controlled by a secretion of hormones from the pituitary, thyroid, adrenal cortex, parathyroids, and male and female sex glands which is so accurately balanced as to be thrilling in its interest Of the hormones elaborated by the pituitary, four are concerned with the development of bone growth hormone, with its direct stimulation of skeletal growth, thyrotrophic hormone, which increases thyroid activity and accelerates growth in the young (though it may cause negative calcium balance and osteoporosis in the adult), gonadotrophic hormone, which stimulates secretion of testosterone and thus augments growth in the young (though androgens and œstrogens cause premature union of epiphyses), and adrenocorticotrophic hormone, which in its final effect inhibits bone growth Growth hormone is secreted by eosinophilic cells of the pituitary, and gonadotrophic hormone by the basophilic cells. Tumours or hypertrophy of the eosinophilic cells causes gigantism in the child and adolescent, and acromegaly in the adult, hypopituitarism from destruction of the anterior lobe causes pituitary dwarfism The hypothyroidism of juvenile myxœdema and cretinism causes dwarfing, whereas hyperthyroidism accelerates skeletal growth Hypogonadism not only suppresses development of the secondary sex characters but also postpones skeletal maturity so that epiphyseal growth continues and the limbs are long, whereas hypergonadism accelerates maturation and by premature closure of the epiphyses causes diminution of stature Hormones of the adrenal cortex inhibit osteogenesis and epiphyseal growth, and in Cushing's syndrome where there is increased pituitary-adrenal function there may be complete cessation of growth and widespread osteoporosis Hyperparathyroidism stimulates osteoclastic activity to such a degree that there is generalised destruction and fibrosis of bone with deformity and multiple fractures

Vitamin control of bone growth—Bone growth is controlled also by vitamins which have been described as "extrinsic hormones." *Vitamin A*

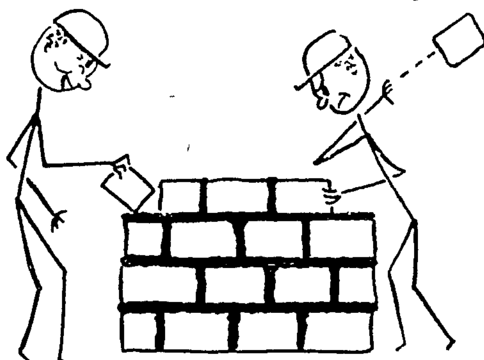


Fig. 592

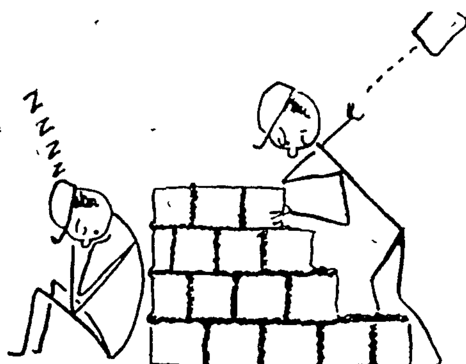


Fig 593

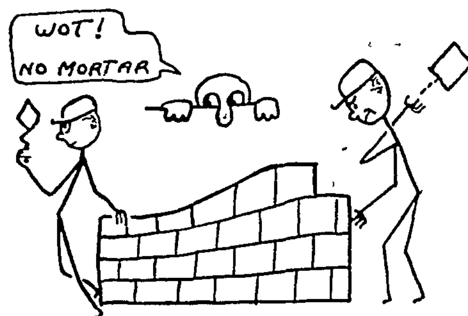


Fig. 594

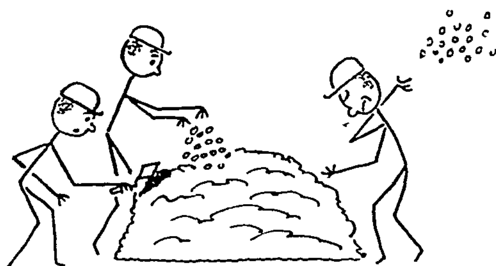


Fig 595

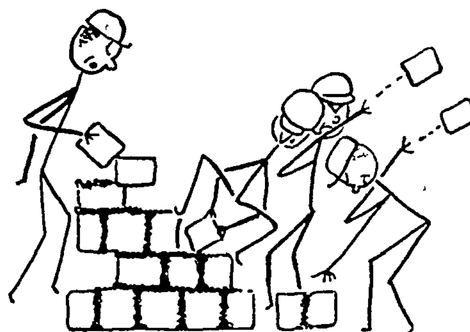


Fig 596

Normal bone is depicted as a brick wall on which two men are at work—a bricklayer on the left and a demolisher on the right. In osteoporosis the bricklayer has gone to sleep. In osteomalacia he is laying bricks without mortar. In osteogenesis imperfecta there is an extra builder, but he is pouring in sand. In hyperparathyroidism there are extra demolishers. *(The cartoons were devised and drawn by Dr Fred C Preston of the Toronto General Hospital to whom thanks are acknowledged.)*

promotes endochondral growth, but excess of this vitamin stimulates osteoclastic as well as osteoblastic activity and the net result may be osteoporosis and pathological fracture Of the twelve or many more vitamins included in *vitamin complex B*, nicotinic acid has proved action in stimulating and accelerating callus formation in fractures *Vitamin C* also stimulates osteoblastic activity, a lack of it causes scurvy with subperiosteal hæmorrhage and epiphyseal displacement, and bone growth is arrested *Vitamin D* promotes absorption of calcium from the alimentary tract, and deficiency causes rickets in the child and osteomalacia in the adult ; the massive seams of osteoid fail to calcify or to be converted into bone Conversely, excess of vitamin D raises the serum-calcium level and causes metastatic calcification in viscera, blood vessels, joint ligaments and the middle ear, sometimes causing ankylosis of joints, blindness and deafness.

Summary of normal bone growth—The stages of growth, development and maintenance of normal bone tissue may now be summarised primitive mesenchymal cells are differentiated as osteoblasts which lay down bone, and osteoclasts which remove it, bone is deposited in the developing embryo on preformed cartilage or membranous condensations of mesenchyme ; continued growth depends upon endochondral ossification at epiphyses, and appositional growth beneath the periosteum ; osteocytes are incapable of mitosis and bone survives only by continued resorption and replacement , thus endosteal ossification continues throughout life , osteoblastic replacement depends upon functional demands , osteoclastic resorption is under hormonal control , bone is almost the sole repositor of calcium and from it the level of serum-calcium must be maintained , bone gives up its calcium if there is failure of absorption from the gut or excessive loss from the kidneys , new bone is first laid down by osteoblasts as osteoid, which is the organic basis of immature bone , osteoid is calcified under the influence of alkaline phosphatase to form immature coarse-fibred woven bone , immature bone is then removed and replaced by mature lamellated bone ; the initial formation of woven bone with later replacement by mature bone is a recapitulation of phylogenetic history , woven bone of early vertebrates was replaced by lamellated bone of mammals—and so it is now in the human The sentence is complete, and the reader is no doubt as breathless as I am—I hope only that he is not also bewildered

Summary of types of bone destruction predisposing to fracture—Weakness of bone predisposing to pathological fracture may arise from developmental or nutritional disturbances, from destruction by infections or tumours, or from neurotrophic dystrophies Nutritional and hormonal disturbances fall into the well-defined groups of osteoporosis, osteomalacia, osteogenesis imperfecta and osteitis fibrosa cystica (Figs 592-596) The maintenance of normal bone may be compared to a brick wall with one workman laying bricks and one removing them In osteoporosis, whether from disuse or from senile change, removal of bone continues but no new bone is laid down (Fig 593) In osteomalacia and rickets from vitamin deficiency or lack of calcium absorption, bricks are laid without good mortar—osteoid is not calcified or converted to mature bone (Fig 594) In osteogenesis imperfecta many osteoblasts lay down imperfect bone (Fig 595) In osteitis fibrosa cystica from hyperparathyroidism bone is laid down at a normal rate but it is removed still more rapidly (Fig. 596).

DEVELOPMENTAL DISORDERS OF BONE CAUSING PATHOLOGICAL FRACTURE

Within recent years we have begun to understand the importance of embryonic environment in the origin of congenital abnormality. It is now recognised that injury to embryonic cells at certain stages of development causes congenital anomaly and, moreover, that when genetic damage has once been sustained it may be transmitted to succeeding generations[1]. Familial and hereditary disorders are sometimes initiated by single incidents such as rubella suffered by the mother during early months of pregnancy,[2,3] or maternal deficiency of vitamins at a time of rapid development of the fœtus. We are only approaching the fringes of this fascinating study, and much more is to be learned before developmental disorders of bone growth can be controlled and prevented, but order is beginning to appear from chaos

Congenital defects of bone tissue—Congenital disorders of the growth of bone tissue, as opposed to failures of epiphyseal development and modelling of individual bones, are typified in osteogenesis imperfecta and osteopetrosis. In *osteogenesis imperfecta* there is genetic damage to primitive mesenchymal cells so that although osteoblasts are differentiated in large numbers they are imperfect in their function, and the development of mature bone is retarded. In *osteopetrosis* genetic damage is seen in the impaired activity of osteoclasts, with failure of resorption of bone causing abnormal thickening and density. In some respects osteopetrosis is the counterpart of osteogenesis imperfecta, but in both conditions—whether the bones are stout or slender, sclerotic or porotic—the bone is immature, fragile and susceptible to fracture. In *arachnodactyly* there is a less-pronounced disturbance in the function of osteoblasts, but pathological fractures are sometimes sustained

Congenital condensing bone dystrophies—Condensation of bone, which is so striking a feature of osteopetrosis, is also observed in other developmental bone diseases but without sufficient similarity to suggest a common pathology, and without special tendency to fracture. *Melorheostosis* is characterised by dense condensation on one side of several bones of a limb. The distribution has been compared to the flow of candle-grease (Figs. 597-599). The bones are not fragile and there is no especial susceptibility to fracture. *Osteopoikilosis* is hereditary and characterised by many dense spots in the bones which have no particular clinical significance. In *punctate epiphyseal dysplasia* or "stippled epiphyses" there are multiple areas of condensation in the epiphyses, often with premature epiphyseal fusion, but the bones are thick and not fragile *Progressive diaphyseal dysplasia* (Engelmann's disease or osteopathia hyperostotica sclerosans) is characterised by sclerosis of the diaphyses but not of the epiphyses or metaphyses of long bones. There is periosteal and endosteal thickening by dense non-trabeculated bone In later stages of the disease osteoclastic and osteoblastic activity are increased and the dense bone is replaced by cancellous bone.

[1] Duraiswami, P K "Insulin-induced Skeletal Abnormalities in Developing Chickens" *Brit med J*, 1950, 2, 384 and 1002, and McFarland, B, *J Bone Joint Surg*, 1951, **33-B**, 45

[2] Gregg, N McA "Congenital Defects in Infants following Maternal Rubella" *Trans Ophthal. Soc Austral*, 1941, 3, 35 *Ibid*, 1944, 4, 119 *Med J Austral*, 1945, 1, 313

[3] Mann, I "Embryological Observations on Congenital Cataract associated with Rubella in the Mother" *Trans Ophthal Soc Austral*, 1944, 4, 115



FIG 597



FIG 598



FIG 599

Melorheostosis

There is dense condensation of bone on one side of the limb, in this case on the lateral aspect of the femur and tibia "flowing like candle-grease" This case is unusual, being bilateral and symmetrical. (*R A F Orthopædic Service*)

during the first few years of life, or in the *adolescent type* where for some years there appears to be no abnormality but four or five fractures are then sustained from trivial injury (osteogenesis imperfecta tarda) Such distinctions between prenatal and postnatal—or fœtal, infantile and adolescent types—are differences only of degree

If the child survives, the tendency to fracture always becomes less in late adolescence and adult life Milk teeth are imperfectly calcified and partly translucent, but permanent teeth are usually normal in appearance Very

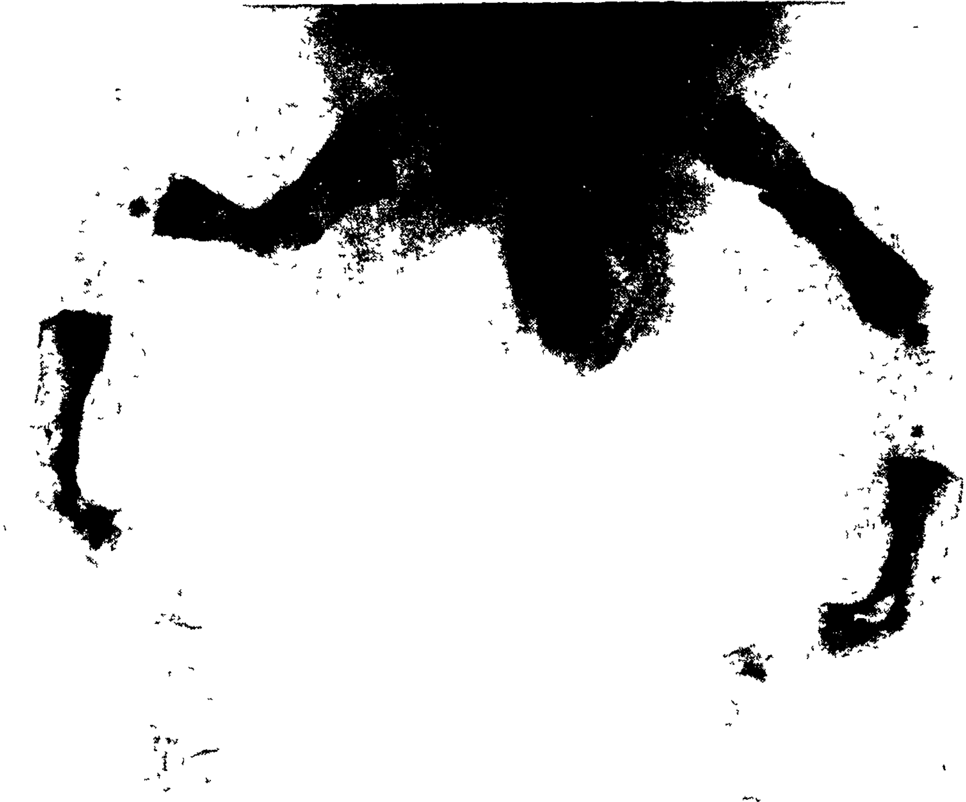


FIG 600

Osteogenesis imperfecta

Fœtal thick-boned type of osteogenesis imperfecta in a still-born child with multiple fractures of all long bones (*Radiograph of specimen in the Pathological Museum of the University of Liverpool*)

often the sclerotics are unduly translucent and appear blue, and members of affected families who do not themselves sustain fractures may nevertheless show blueness of the sclerotics—a deep indigo, easily distinguished from the smoky-blue eyes of normal babies and the Irish eyes of some adults (Figs 601-602) Otosclerosis may develop in patients who live to the third decade; fragility of bone, blue sclerotics and otosclerosis constitute Van der Hoeve's syndrome. Apart from the tendency to fracture of bone there is laxity of ligaments and susceptibility to joint strain and dislocation.

Three pathological types of osteogenesis imperfecta may be distinguished.

Thick-boned type—In severe prenatal cases the shafts of long bones are usually much thicker than normal (Figs 603-604) Genetic damage to primitive

mesenchymal cells is such that although large numbers of osteoblasts are differentiated, and much osteoid and immature bone is laid down, conversion to mature bone is delayed or arrested The bones are thick but they are fragile *Thin-boned type*—In infantile and adolescent types the long bones are usually slender and there is porosis of the vertebral bodies, which become thin and biconcave. The cortex is thin and bone structure shows diminished density, sometimes with honeycombing. *Osteogenesis imperfecta cystica*—In a very few cases honeycombing is so generalised as to suggest a subtype—osteogenesis imperfecta cystica.[1 2]



Fig 601



Fig 602

Osteogenesis imperfecta

Mother and daughter with osteogenesis imperfecta showing indigo-blue coloration of the sclerotics Members of every generation of this family suffered multiple fractures from congenital bone fragility

Treatment of fractures in osteogenesis imperfecta—It has already been emphasised that despite genetic deficiency of mesenchymal cells there is an abundance of osteoblasts with vigorous growth of osteoid and coarse-fibred bone Fractures always unite promptly and soundly, and sometimes, indeed, there is excess of callus The tragedy is that many doctors, taking pride in recognition of the syndrome, have coupled with it a belief that deformity is inevitable, patients have often been left in bed without the splints that would otherwise have been applied There is no reason why the principles of fracture treatment should be applied less faithfully when a

[1] Fairbank, H A T *Proc Roy Soc Med*, 1935, 28, 1611
[2] Fairbank, H A T *J Bone Joint Surg*, 1948 30-B, 180



Fig. 603

Fig 604

Osteogenesis imperfecta

Radiographs of the femur and tibia of the patient shown in Figure 602 She sustained twenty-eight fractures in twenty-five years—but it is to be noted that the bones are now dense At the age of twenty-five years the patient's susceptibility to fractures has almost disappeared

child sustains twenty fractures than when he sustains one. In this disease the tendency to fracture often disappears after adolescence, and the density of bone may even be increased (Figs. 603-604). When approaching the age of twenty the patient may be ready to pursue an almost normal life, provided only that he is not already so deformed and dwarfed as to be incapable of standing or walking. When a fracture is sustained, alignment at least should be maintained, particularly in the lower limb, either by simple local splinting or by skin traction in a Thomas' splint. It is possible that intramedullary nailing may help to solve the problems not only of treating recent fractures but of protecting the patient from future fractures.

Hyperplastic callus formation in osteogenesis imperfecta.—In the fractures of osteogenesis imperfecta callus formation is usually plentiful and sometimes excessive, but the excess is absorbed when the fracture consolidates. Occasionally tremendous excess of callus formation develops around the shaft of a fractured long bone, particularly the femur, persisting as permanent thickening even up to three or four times the normal dimension of the bone. This may occur when there is no clear evidence of a preceding fracture.[1] The tissue shows many islands of cartilage and it has been described by Baker as "chondrosteoid tissue," intermediate between cartilage and woven bone. Not only does the new bone fail to resorb and consolidate but, unlike traumatic subperiosteal ossification and the subperiosteal ossification of scurvy (with which it was confused by Brailsford[2]), it tends to invade adjacent atrophic muscle fibres. The distinction from sarcoma may be difficult.

Osteopetrosis generalisata (marble bones, Albers-Schonberg's disease, osteosclerosis fragilis generalisata).—Osteopetrosis has a strong familial tendency and is sometimes inherited. There is genetic deficiency of osteoclastic resorption, and the metaphyseal region of long bones shows club-like thickening with remarkable increase in bone density (Figs. 605-609). There



FIG. 605

Osteopetrosis generalisata

There is generalised density of bone and metaphyseal thickening. (See Figures 606-609.)

is increase in the number and thickness of trabeculæ with a disordered irregular architecture and complete failure of modelling resorption, so that continued periosteal apposition and surface thickening are accompanied by failure to develop a medullary cavity. Consequent reduction in the blood-forming marrow causes aplastic or leukæmic anæmia which may be fatal. The abnormal growth of bone sometimes continues uninterruptedly until the

[1] Fairbank, H. A. T., and Baker, S. L. "Hyperplastic Callus Formation, with or without Evidence of Fracture, in Osteogenesis Imperfecta." *Brit. J. Surg.*, 1948, 36, 1.
[2] Brailsford, J. F. *Brit. J. Radiol.*, 1943, 16, 129.



Fig 606

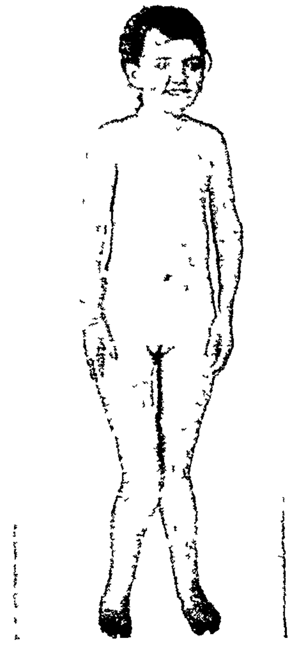


Fig 607

Fig 608

Osteopetrosis generalisata

There is abnormal density of all bones with club-like thickening of the metaphyses, encroachment on the medulla causing anæmia, and constriction of cranial foramina with paralysis of the facial and auditory nerves

epiphyses fuse, and thereafter the dense and thickened skeleton remains unchanged; but often there is a curious periodicity, with intermissions or even complete cessation of the developmental error, so that metaphyses show alternating bands of dense and normal bone The vertebral bodies may show dense layers above and below, with a less dense layer between, in the pelvis there are curves of dense bone parallel to the iliac crests, the carpal and tarsal bones show circular distribution of dense bone surrounding a clear centre, or a dense centre surrounded by less opaque bone or perhaps several concentric rings of dense and porous bone Not only does encroachment of the sclerosed bone on the marrow cause anæmia, but constriction of the cranial foramina may give rise to optic atrophy, facial palsy, or other cranial nerve lesions. The bones are sometimes intensely hard like marble but in other cases they are more chalk-like in consistency and are susceptible to fracture. When fractures occur they are usually sharp and abrupt, in a



Fig 609

Osteopetrosis generalisata

Same case as that shown in Figures 605-608 There are united fractures of the shafts of both femora

strictly transverse axis corresponding to one of the planes of disordered epiphyseal growth The patient shown in Figures 605-609 sustained symmetrical fractures of both femora Another patient sustained ten fractures in twenty years Callus formation is sometimes slow but fractures always unite quickly and consolidate soundly

Arachnodactyly is a rare developmental error of growth in which general tallness of build is associated with slenderness of the limbs and abnormal lengthening, particularly of the phalanges of the hands and feet (Figs 610-612) There is flexion contracture of the fingers and toes, and bilateral coxa vara with central protrusion of the acetabula The bones have a thin cortex with widely separated trabeculæ and poor calcium content, and pathological fractures are not uncommon [1-3]

[1] Burrows, H J "The Bone Dystrophies," in "Modern Trends in Orthopædics," edited by H Platt London Butterworth & Co (Publishers) Ltd, 1950, 437
[2] Ellis, R W B "Four Cases of Fragilitas Osseum with Blue Sclerotics" *Proc Roy Soc Med*, 1931, 24, 1054
[3] Kersley, G D "Fragilitas Osseum and Allied Conditions" *St Bart's Hosp Rep*, 1935, 68, 159



Fig 610

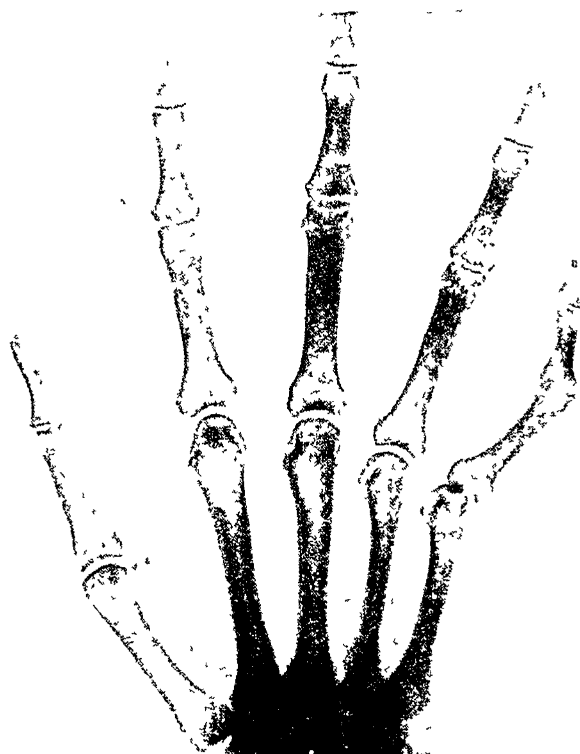


Fig 611



Fig 612

Arachnodactyly

The "spider hand" and "spider foot" of arachnodactyly are shown in the tremendous lengthening of phalanges metacarpals and metatarsals In this case there is thickening of the shaft of the second metatarsal flexion contractures of fingers and toes, and central protrusion of the acetabula The long slender bones are susceptible to fracture *(Case presented at Staff Conference, London Hospital by Mr F G Ward of Ashford)*

Dyschondroplasia (Ollier's disease, or multiple enchondromatosis) is characterised by such delayed and imperfect conversion of epiphyseal cartilage to bone that the metaphyses include a considerable proportion of unossified cartilage and are often expanded diffusely by enchondromata, but without exostoses as in diaphyseal aclasia The growth disorder is seldom generalised —it may affect the epiphyses of a single bone, of several bones of a single limb, of both lower limbs, or of both limbs on one side of the body Moreover, it does not usually affect every part of an epiphysis equally Sometimes there is normal growth on one side of an epiphysis with areas of unconverted cartilage and delayed ossification on the other In the knee joint, for example, genu valgum may develop from greater involvement of the lateral than the medial part of the epiphysis Very often some columns of cartilage



FIG. 613

Dyschondroplasia

Typical changes in the hands in dyschondroplasia. At every epiphysis there is delayed conversion of cartilage to bone. Secondary calcification of the cartilage is seen in the radial and ulnar metaphyses

cells ossify normally, whereas adjacent columns remain unossified, thus accounting for the longitudinal striation of metaphyses seen in radiographs. The striæ of reduced density, representing columns of unconverted cartilage, are based at one end on the epiphyseal plate and are lost at the other end in the shaft of the bone In the ilium unossified sectors of cartilage based on the epiphysis at the crest, alternating with areas of more normal bone, give the typical radiographic appearance of a fan which may be a diagnostic sign in doubtful cases (Fig 614) Even when there is limited involvement of an epiphysis, growth is usually retarded and there is dwarfing or inequality of limb length, sometimes with spontaneous dislocation If the radius alone is affected, the normally growing ulna dislocates at its lower end, and conversely if the ulna is more affected than the radius, and is proportionately shorter, the shaft of the radius curves and the upper end may dislocate When there is still a considerable proportion of normal bone in the metaphysis,



FIG 614
Dyschondroplasia
Typical fan-like ossification of the ilia in dyschondroplasia (*From Sir Thomas Fairbank's article in "J Bone Joint Surg,"* 1948, 30-B, 697)



FIG 615

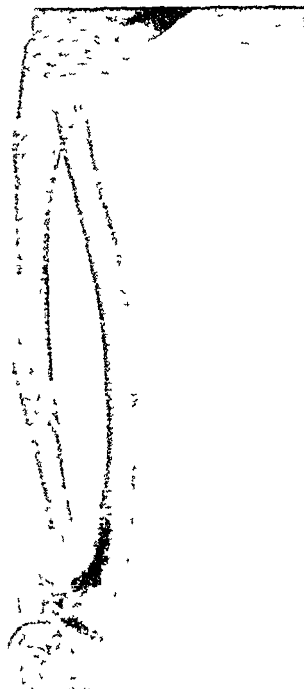


FIG. 616

Dyschondroplasia
Boy aged eleven years with dyschondroplasia causing arrested growth of the back of the lower femoral epiphysis and the front of the upper tibial epiphyses A fracture of the ulna showed delayed union with refracture from trivial injury, consolidation was unsound even six months later (Fig 616)

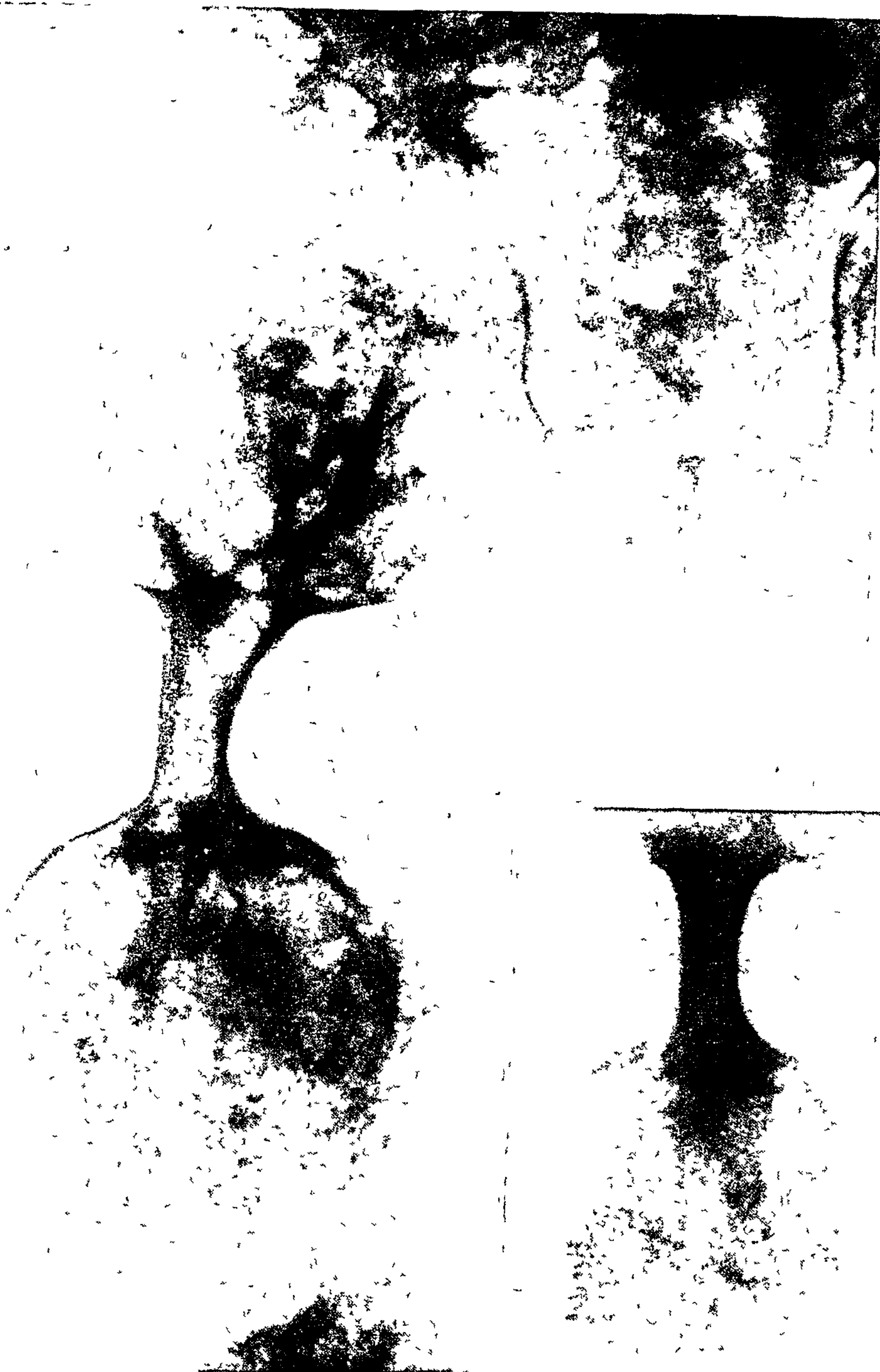


Fig 617 Fig. 618

Dyschondroplasia

When all parts of the epiphyses are involved equally in arrested conversion of cartilage to bone, the metaphyses become widely expanded with masses of hyaline cartilage which are fragile despite secondary calcification In this case there was a fracture at the junction of the metaphysis with the femoral shaft (see inset) It united soundly

fractures are uncommon; but when a fracture is sustained union is often slow and delayed (Fig 616). If every part of an epiphysis is involved, so that the metaphysis is expanded by a mass of pure hyaline cartilage, fractures are more frequent, despite the misleading appearance of density often observed from secondary calcification of the enchondroma (Figs 617-618)

The cause of dyschondroplasia is obscure. There seems to be no hereditary or familial influence, but several features—notably the curiously selective rather than generalised involvement of epiphyses, the normal blood chemistry, and the occasional association with congenital hæmangiomata (Maffucci's syndrome)[1]—support the view that it is the result of a primary germ cell defect. It has recently been suggested that dyschondroplasia is in fact a form of vitamin-resistant rickets[2] Certainly infants and children with inherited resistance to vitamin D may develop general skeletal deformity with dwarfing and epiphyseal disturbance resembling some types of dyschondroplasia, and they respond to massive doses of vitamin D for which they have an astonishing tolerance—but this is to say no more than that vitamin-resistant rickets has sometimes been mistaken for dyschondroplasia Much more evidence is needed before we can agree with Compere that "dyschondroplasia is in reality a vitamin-D-resistant type of rickets"[3]

NUTRITIONAL AND VITAMIN DEFICIENCIES CAUSING PATHOLOGICAL FRACTURE

Pioneer work on vitamins, first pursued in England by Mellanby and Bourne, has led to the virtual disappearance in many countries of scurvy and rickets In Britain and America more than twenty years have passed since epiphyseal separation, subperiosteal hæmorrhage and "pseudo-paralysis" have been seen from scurvy, or gross deformity and dwarfing, with inability to stand or walk, from rickets. Nevertheless these nutritional deficiencies still cause pathological fracture in some parts of Africa, India and Asia. Moreover, interest has recently been aroused in minor deficiencies of vitamin C and the "subscurvy state" in relation to the healing of fractures, and in various forms of vitamin-resistant rickets

Vitamin C and scurvy—Deficiency of vitamin C, which is in highest concentration in citrous fruits and leafy vegetables, was formerly the dreaded peril of long sea voyages, sieges of war and polar explorations. Scurvy is characterised by extensive hæmorrhage in muscles and subperiosteal planes, swelling and bleeding of the gums with loosening of teeth, and failure of wounds to heal normally. In growing children there is osteoporosis, fibrosis of marrow, and epiphyseal separation or fracture Vitamin C is needed for the growth and maintenance of collagen matrix and, if there is deficiency, the cells responsible for the elaboration of intracellular substance are not differentiated and may revert to fibroblasts The third zone of the epiphyses is widened. Preparatory calcification proceeds normally but osteoid is not laid down, or at best the osteoblasts produce a homogeneous matrix with little collagen content. Fractures often occur at the costo-chondral junction of ribs or at the epiphyseo-diaphyseal junction of long bones. There are hæmorrhages because support of the capillary vessels

[1] Carleton, A., Elkington, J, Greenfield, J. G, and Robb-Smith, A. H T. *Quart J. Med*, 1942, N.S. **11**, 203.
[2] Holt, J. F *Amer J. Roentgen*, 1950, 64, 590
[3] Compere, E Editorial, "Year Book of Orthopædics" Chicago Year Book Publishers, 1950.

is weakened by imperfect development of connective tissue around them. For the same reason, healing of wounds is delayed Ascorbic acid is needed for the growth of collagen in repairing wounds of soft tissue, as well as for the growth of osteoid in uniting fractures of bone.

The subscurvy state—The relationship between vitamin C and the adreno-cortical hormones, which also control growth of collagen, and the wider implications of this vitamin–hormone balance, were discussed on page 231 Apart from its direct influence on the healing of wounds of connective tissue, ascorbic acid is needed to support the potential of the adrenal cortex in defence against cold, fatigue and other stresses which initiate the adaptation syndrome. If the intake of ascorbic acid is insufficient, the adrenal cortex becomes hypertrophied in its endeavour to meet the demand, and it soon loses all powers of defence The optimal daily intake of this vitamin may be estimated at 50 mgm. Many individuals receive far less than this without usually showing ill effect, but they need much more when stresses are to be met There is little doubt that deficiency of ascorbic acid, or the "subscurvy state," has often passed unrecognised as a cause of slow union of fractures, delayed healing of wounds and lessened resistance to shock After severe injury, especially when coupled with exposure, fatigue or exhaustion, saturation should be achieved by giving 300 mgm. three times daily for three days, with a maintenance dose thereafter of not less than 50 to 100 mgm. daily.



FIG 619

Typical rachitic metaphysis with epiphyseal widening and uncalcified osteoid

Rickets—The vitamin D content of egg yolk and dairy products in an ordinary diet is low, but there is us y sufficient provitamin D, cholesterol and ergosterol, to be converted b ultraviolet irradiation Cholesterol stored in the skin where it is changed to vitamin D by sunlight, and main cause of deficiency is insufficient exposure of the body to light Th function of this vitamin is the regulation of calcium-phosphorus metabo primarily by promoting absorption of calcium from the alimentary tract By tending to increase the blood-calcium level, parathyroid function i depressed and the urinary output of phosphorus is reduced

The effects of deficiency of vitamin D may therefore be summarised absorption of calcium from the gut is reduced, calcium remaining in t alimentary tract combines with phosphorus to form insoluble calcium phosphate so that phosphorus absorption is also reduced; reduced calc intake stimulates parathyroid secretion and the glands hypertrophy, that although the level of blood-calcium remains normal, calcium is no deposited in bones and it may even be withdrawn from them; parathyroi



Fig. 620

Fig. 621

Rickets

Indian child, aged three years, who has never walked. The epiphyses are wide and there is evidence of imperfectly calcified osteoid laid on the shafts of the femora. In one femur there is an un-united fracture of the lower shaft, and a recent fracture of the middle third. Fractures of the shafts of both forearm bones are uniting (*By courtesy of Dr Mungo-Thompson, King Edward VIII Hospital, Durban*)

hyperactivity increases phosphorus output in the urine, and the ... phosphorus level is therefore low ; increasing osteoblastic activity produc... uncalcified osteoid raises the level of serum-alkaline-phosphatase The bloo... chemistry of rickets is therefore characterised by a normal serum-calci... low blood-phosphorus, and raised alkaline-phosphatase

In growing children the first consequence of vitamin D deficiency ... failure of calcification at the epiphyses The zone of proliferating cartila... cells becomes progressively deeper and osteoid remains uncalcified. Resistanc... of uncalcified bone to modelling resorption causes widening and broa... of epiphyses, with the characteristic "rachitic metaphysis" of long bon... (Fig. 619) and "rachitic rosary" of ribs. Similarly, in the shafts of lon... bones, appositional growth of osteoid remains uncalcified and unconverted to mature bone. The bones may be thick but they are weak and then become deformed Incomplete crack fractures develop on the convex side of the



FIG. 622
Patient, now aged nineteen years, with vitamin-resistant rickets (see Figs. 623-624)

bent bones, and trivial injury may convert any of these cracks into a complete fracture (Figs. 620-621) The effect of giving adequate supplies of vitamin D is surprisingly prompt. There is at once calcification of osteoid with conversion to mature bone, restoration of the epiphyses to a normal size, and hardening of the bones with disappearance of the tendency to pathological fracture If the dosage of vitamin D is too high, metastatic calcification may occur in the kidneys, bronchi, alveoli of the lungs, mucous membrane of the stomach, blood vessels, joint ligaments and middle ear.

Vitamin-resistant rickets—In some children and adolescents, even when there is no disease of the renal or alimentary tracts, rickets fails to respond to vitamin D in usual dosage, or to ultraviolet irradiation of the skin The blood chemistry is the same as in ordinary rickets—normal serum-calcium, low serum-phosphorus, and raised serum-alkaline-phosphatase—but the serum-phosphorus remains low despite vitamin therapy. Severe deformities



Fig. 623



Fig. 624

Vitamin-D-resistant rickets

Radiograph of patient shown in Figure 622 who had evidence of rickets from infancy Treatment by cod-liver oil and sunlight were of no avail Severe deformities developed with pathological fractures of the tibiæ, femora and forearm bones Serum-calcium 11 5 mgm , serum inorganic phosphate 3 mgm , blood urea 35 mgm , total fat in stool within normal limits. had been unable to stand or walk before the age of fifteen, when these radiographs were taken After treatment by a quarter-million units of calciferol daily the disease was arrested and the fractures united The deformities are now being corrected. (*Presented at Staff Conference, Robert Jones and Agnes Hunt Orthopædic Hospital, investigated at the Victoria Infirmary, Newcastle-on-Tyne*)

develop and there may be multiple pathological fractures (Figs 622-624). Vitamin resistance is believed to be congenital and sometimes familial Success has been reported from the administration of spectacularly high doses of the vitamin, even up to one and a half-million units daily.[1] Resistance to small dosage is reflected in tolerance to high dosage, but nevertheless there must be careful watch for signs of hypervitaminosis D

Cœliac rickets (idiopathic steatorrhœa, non-tropical sprue)—In cœliac disease of children, and idiopathic steatorrhœa of adults, there is deficiency of pancreatic ferments causing incomplete digestion and absorption of fats. The stool is bulky and pale, with an excess of both split and unsplit fats. The resulting formation of calcium soap and the generally accelerated flow through the alimentary tract prevent absorption of calcium and phosphorus, and there may even be tetany. As in true rickets, the serum-calcium is normal or low, serum-phosphorus is low, and serum-alkaline-phosphatase is high ; and there is epiphyseal thickening, general osteomalacia with deformity, and pathological fracture from trivial injury. So complete is the failure of calcium absorption that fractures unite very slowly. The fat intake should be reduced to 50 or 60 grammes a day Hydrochloric acid may be given by mouth to relieve the associated achlorhydria. Calcium gluconate by mouth and injection, massive doses of vitamin D, and ultra-violet irradiation are advisable Success has been claimed recently from the administration of folic acid (vitamin M) in dosage of from 10 to 100 mgm. daily [2]

Renal rickets (renal infantilism, renal dwarfism, renal osteitis fibrosa cystica, renal acidosis from tubular insufficiency, Fanconi syndrome)—In this group of renal diseases there is failure of epiphyseal calcification and delayed growth with widening of epiphyseal plates, thickening of metaphyses, and formation of much uncalcified osteoid, exactly as in true rickets Coxa vara often develops from separation and displacement of the upper femoral epiphyses, and there may also be deformity at the knee joints—either genu valgum or genu varum The disorder may arise from congenital cystic disease or congenital hydronephrosis causing total renal insufficiency, glomerular as well as tubular, or from tubular insufficiency alone which may be of two types : 1) Fanconi's syndrome in which the tubules fail to reabsorb phosphates, glucose and many amino-acids so that there is low serum-phosphorus, renal glycosuria and amino-aciduria ; 2) renal acidosis in which renal failure to make ammonia and excrete an acid urine depletes the serum of phosphorus In each syndrome there is hypercalcuria causing renal calcinosis or lithiasis The prognosis is grave Improvement may sometimes be gained by treatment with sodium salts and massive doses of vitamin D.

Osteomalacia (hunger osteomalacia, puerperal osteomalacia, vitamin-D-resistant osteomalacia, cœliac osteomalacia, renal osteomalacia)—Osteomalacia in the adult is the counterpart of rickets in the child—the causes are identical, blood chemistry the same, and bone changes similar.

Hunger or famine osteomalacia was studied in Vienna after the first world war, mostly in elderly women By reason of insufficient intake of calcium and vitamin D the bones became porotic and there was formation

[1] Albright, F, Butler, A. M, and Bloomberg, E "Rickets resistant to Vitamin D Therapy" *Amer J. Dis. Child*, 1937, 54, 529
[2] Spies, T D *Lancet*, 1946, 2, 225 *J Amer med Ass*, 1946, 130, 474



FIG 625





FIG 626

Cœliac osteomalacia causing fractures

For nearly fifty years this patient suffered from sprue and she sustained fractures of the ischial ramus, pubic ramus, neck of the femur, twelfth dorsal vertebra, and first lumbar vertebral body All vertebral bodies show a tendency to bi-concavity from pressure of the elastic intervertebral discs There is scoliosis from lateral crushing The shaft of the femur is obviously porotic, with loss of normal trabeculation and thinning of the cortex.

of uncalcified osteoid with fibrosis of the marrow. The level of blood-calcium and blood-phosphorus was low—sometimes with Chvostek's sign, cramps and even tetany There was bone tenderness, kyphotic deformity with bi-concave vertebral bodies, ballooned intervertebral discs, and often spontaneous fracture of the rarefied long bones

Puerperal osteomalacia arises from failed absorption of calcium by reason of dietetic insufficiency and lack of exposure to sunlight, aggravated by the drain of calcium needed for the fœtal skeleton and still more by the considerable loss of calcium from the lactating breast. The level of blood calcium may be so low as to cause tetany. The malacic bones are tender and susceptible to deformity—notably triradiate compression of the pelvis. There may be pathological fracture of the shafts of long bones

Vitamin-resistant osteomalacia arises in well-fed, apparently healthy women, the condition corresponding exactly to vitamin-D-resistant rickets. Often there are multiple spontaneous symmetrical fractures—the condition described as Milkman's syndrome The level of blood phosphorus is low and there is a negative calcium and phosphorus balance Ultraviolet irradiation and ordinary dosage of vitamin D has no effect, but there is a prompt response to massive dosage, as high as half a million or even one million units daily. *Cœliac osteomalacia*—Steatorrhœa or sprue causes osteomalacia by reason of failed absorption of calcium from the alimentary tract The bone change is most evident in the spine where spontaneous crush fractures may be sustained, and there are sometimes fractures of long bones. *Renal osteomalacia*—Renal tubular insufficiency in the adult with continued loss of phosphate from the kidney may cause osteomalacia in the adult exactly as it causes renal rickets in the infant

Milkman's syndrome (multiple spontaneous idiopathic symmetrical fractures)—Rarefaction of bone in Milkman's syndrome arises from osteomalacia of the types already discussed.[1] The multiple spontaneous " fractures " which often occur symmetrically in both lower limbs, or both upper limbs, are in fact transverse bands of uncalcified osteoid These bands of malacic bone have been described as pseudo-fractures, *umbauzonen*, or Looser's transformation zones They are not true fractures, and they disappear when the cause of osteomalacia is removed

HORMONAL IMBALANCE CAUSING PATHOLOGICAL FRACTURE

The control of bone growth by hormones of the pituitary, parathyroid, adrenal and sex glands has already been outlined Discussion is difficult because as yet we know little of the balance and interplay of these various hormones, of the selectivity by which the same hormone has different effects in different animals, of the particular sensitivity to hormonal influence of certain bones such as the vertebræ in humans and the pelvis in rats, or of the specific influence of hormones on different types of ossification—for example, pituitary growth hormone on periosteal ossification, and oestrogens on endochondral ossification For the moment we can do no more than outline the disorders that have proved relationship to recognised syndromes with pathological fracture

[1] Strang, C "The Looser-Milkman Syndrome—Occurrence in a Case of Idiopathic Steatorrhœa." *Brit J. Surg*, 1951, 38, 489

Hyperparathyroidism—In 1888 a certain Herr Bleich fell from a ladder and sustained a fracture of the neck of the femur; soon afterwards he fractured his clavicle, and then the shaft of one femur. He died At

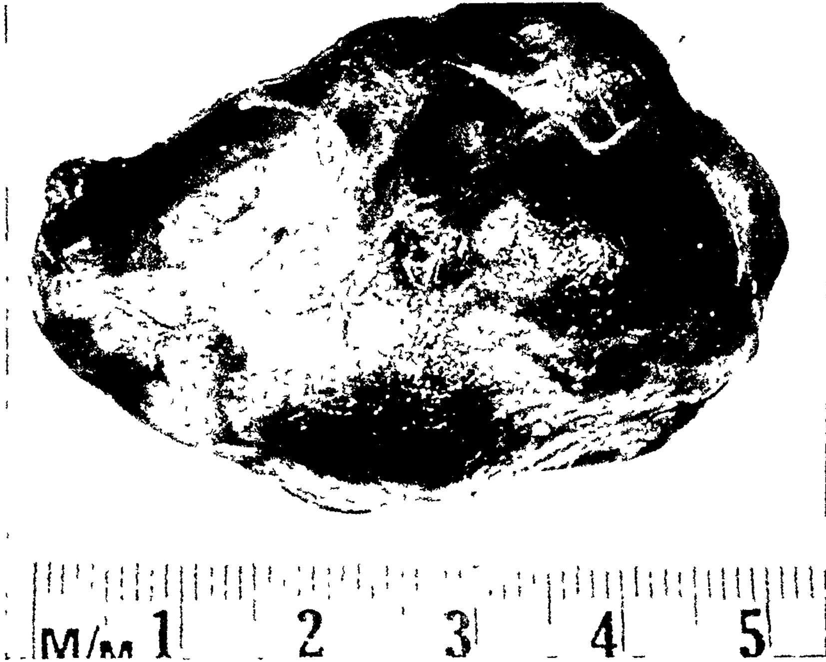


FIG. 627

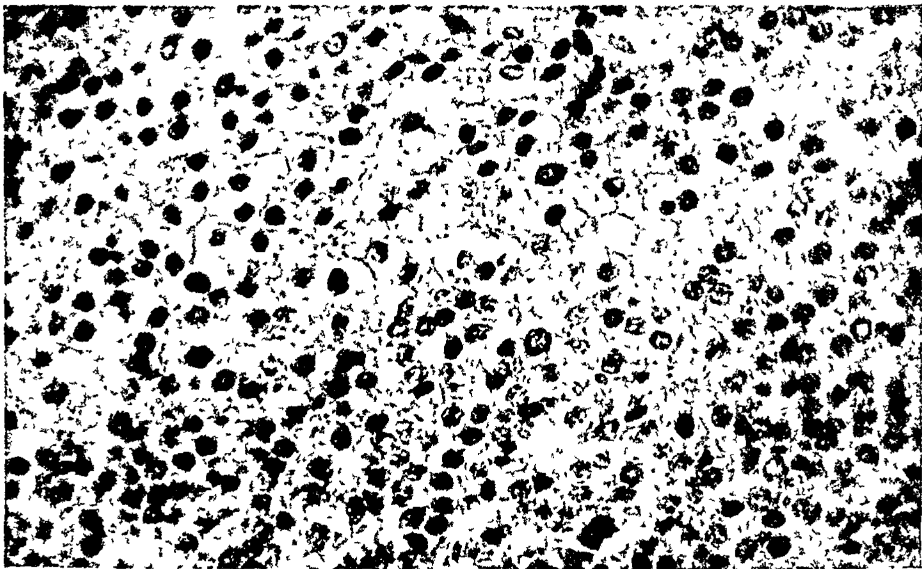


FIG 628

An unusually large parathyroid adenoma which was the primary cause of bilateral fractures of the femoral necks (see Figs 629-632) The histological appearances of the tumour were typical (*Ehrlich's hæmatoxylin and eosin ×340, microphotograph by Mr King, London Hospital, courtesy of Dr Wood and Prof D Russell*)

autopsy von Recklinghausen recorded fibrosis of bone, cyst formation and brown giant-cell tumours, and added that "above the left thyroid gland a lymph gland, red-brown in colour, is present" This was the first record of a parathyroid adenoma with fibrocystic disease and pathological fracture

Increased parathyroid secretion from hyperplasia or neoplasia of the glands mobilises calcium from bone and raises the serum-calcium level. At first it was believed that the hormone acted directly on bone in promoting release of its calcium ; it is now recognised that the influence is less direct and that the specific action of parathormone is on the kidneys, causing increased urinary excretion of phosphorus with consequent lowering of serum-phosphorus, so that calcium is mobilised from bones in order to maintain a constant calcium-phosphorus equilibrium in serum. Lowering



Fig 629



Fig 630

Hyperparathyroidism

A man, aged twenty-six years, turned quickly on a railway platform to greet his wife and collapsed He was admitted to Oldchurch Hospital with fractures of both femoral necks There was obvious porosis of the bones, and study of the case showed that there was hyperparathyroidism from an adenoma (see Figs 627-628 and 631-632)

of serum-phosphorus is therefore associated with raised serum-calcium, the level ranging from 12 to 20 mgm instead of the normal 9 to 11 mgm per cent Hypercalcæmia causes hypercalciuria, and very often there are multiple renal calculi , indeed this complication is so frequent that the investigation of every patient with stones in the kidney should include study of the blood chemistry even if hyperparathyroidism is not suspected on other grounds

The clinical manifestations of hyperparathyroidism are sometimes obvious because there is extensive cystic change in all bones, generalised porosis, osteoclastomatous cysts, peppery granulation of the skull, recurring spontaneous fractures, and renal colic with hæmaturia from calculi [1] Very often, however, the signs are much less obvious and some patients have suffered

[1] Hunter, D , and Turnbull, H M *Brit J Surg* , 1931, 19, 203.

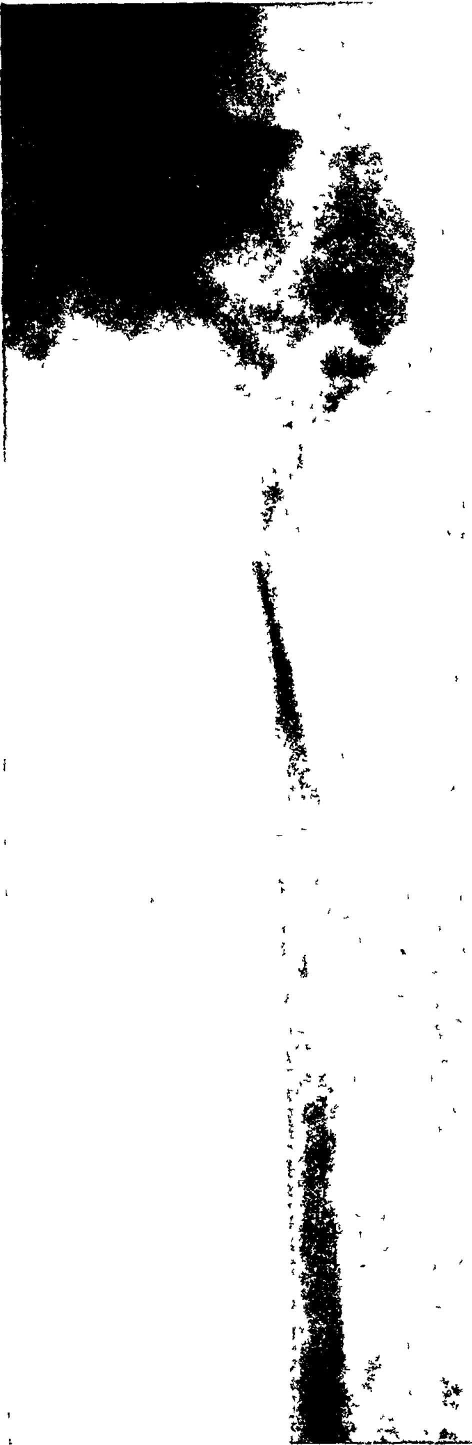


FIG. 631



FIG 632

Hyperparathyroidism

Same case as in Figures 627-630 Porosis of the fractured bones was confirmed by comparing radiographs with films of identical penetration and exposure of a normal subject of similar size and weight (Fig 632) Serum-calcium was 17 9 mgm per cent serum-phosphorus 3 4 mgm The tumour of the parathyroid could be felt It was removed by Mr W. Park (Fig 627) After operation there was tetany, controlled by calcium gluconate injected intravenously and parathormone intramuscularly The femoral fractures were treated successfully by Russell traction

(*Reported by the courtesy of Mr Raymond King, Orthopædic Surgeon, Oldchurch Hospital, formerly of London Hospital*)

for many years before the diagnosis has been established. In one case a limb was amputated for what was believed to be a malignant tumour of bone, and only nine years later was a parathyroid tumour recognised and removed.[1] There may be no more than thickening of one or more bones with tenderness often diagnosed as "fibrositis," an epulis or cyst of the jaw without changes elsewhere in the skeleton, generalised kyphosis with reduction of stature, or perhaps a fracture sustained from trivial injury. Sometimes there are no obvious bone changes: there may be muscle weakness mistaken for myasthenia gravis; polyuria and polydipsia wrongly diagnosed as diabetes insipidus; or stones in the kidney which are not recognised as part of a constitutional disease. Loosening of teeth without pocketing may be the first evidence (but radiographic examination usually shows absence of the lamina dura—the line of bone condensation around the roots of teeth—and Albright goes so far as to say that disappearance of the lamina dura is a specific sign in all cases of hyperparathyroidism[2]).

The first manifestation of hyperparathyroidism may be an osteoclastoma. Punch biopsy shows many giant cells, and even at open biopsy the appearances may seem to be typical of osteoclastoma, but general rarefaction of bones, made obvious by comparing radiographs of the patient with films of identical penetration and exposure of another subject of similar age and size, should call for estimation of the blood calcium and phosphorus. If a fracture of bone is found to be associated with general porosis and subperiosteal apposition of new bone of subnormal density, the surgeon should be on the alert—there may be no extensive cystic change, and there may have been only one fracture, but nevertheless the blood chemistry should be studied. If the serum-calcium is high, serum-inorganic-phosphate low, and serum-alkaline-phosphatase raised, with increased urinary output of both phosphate and calcium, and a negative calcium balance, then the parathyroid glands should be explored.

As a rule there is a parathyroid adenoma which must be removed. Very occasionally an enlarged gland can be felt in the neck (Fig. 627), but the tumour usually lies deeply, and sometimes it is so deep in the superior mediastinum as to escape notice even at operation. One case is recorded where the neck was explored six times before an adenoma was exposed after splitting the sternum.[3] In other cases there is no tumour, but all four parathyroid glands are hyperplastic. Two or perhaps three of the glands must then be removed, care being taken to control post-operative tetany by injecting calcium gluconate and parathormone.

Secondary hyperparathyroidism—Any condition that lowers the serum-calcium value stimulates increased secretion from the parathyroid glands. Thus secondary hyperparathyroidism occurs when there is calcium deprivation, rickets or osteomalacia causing failed absorption of calcium, pregnancy with reduced calcium intake or vitamin deficiency, or any type of renal failure or insufficiency associated with phosphate retention. It is even possible that hyperplasia of the parathyroids in response to low serum-calcium may be the first cause of adenomata of the glands, but this is speculative.

[1] Beck, A. *Arch. klin. Chir.*, 1928, **152**, 123.
[2] Albright, F., and Reifenstein, Jr., E. C. "Parathyroid Glands and Metabolic Bone Disease." Baltimore: The Williams and Wilkins Co. 1948, 57.
[3] Bauer, W., Albright, F., and Aub. *J. clin. Invest.*, 1930, **8**, 229.

Hyperpituitarism (Cushing's syndrome)—Cushing's description of the fat, bearded, diabetic woman is the archetypal picture of pituitary-adrenal hyperfunction. The syndrome may arise from a basophilic adenoma of the pituitary which increases the output of adreno-corticotrophic hormone and therefore stimulates the adrenal cortex, or it may arise from a tumour of the adrenal cortex itself. In either event there is increased secretion by the adrenal cortex of gluco-corticoids (11-oxysteroids, sugar or S-hormones of Albright) which promote the conversion of protein to sugar and are antagonistic to insulin. This catabolic action, causing breakdown of protein with release of glucose, discourages osteogenesis and apposition of new bone. There is also increased adrenal secretion of androgen hormone (nitrogen or



Fig. 633

Cushing's syndrome

Section of fractured rib in Cushing's syndrome showing repair by surrounding callus in which there is much woven bone. There is osteoporosis, but the fracture stimulated growth of osteoid within the bone fragments as well as in the abundant surrounding callus. (*Dr Robb Smith's case, photomicrograph by Mr King, Bernhard Baron Laboratories, London Hospital, courtesy of Prof D Russell, hæmatoxylin and eosin* ×6.)

N-hormone of Albright) which promotes the growth of axillary and pubic hair in adults and causes premature union of epiphyses in children. The fatness of Cushing's syndrome may be a direct result of the secretion of S-hormone with its diabetogenic influence, or it may arise from excessive secretion from the adrenals of fat hormone (Kendall's compound A or compound B); there is often increase in fat without total increase in weight, suggesting protein–fat conversion. The complete clinical picture of Cushing's syndrome from pituitary-adrenal hyperfunction is therefore adiposity of the face, neck and trunk with sparing of the extremities, hypertrichosis and amenorrhœa in women, cessation of bone growth and shortness of stature in children, hyperglycæmia, hypertension, polycythæmia, tendency to bruising and to thinning of the skin, sometimes nephrosclerosis and albuminuria, and osteoporosis with rarefaction of vertebral bodies which become bi-concave

from the pressure of intervertebral discs and easily sustain crush fractures from trivial injury, kyphosis with reduced stature, persistent pain in the back from vertebral deformity, fractures of the ribs and clavicle, and sometimes fractures of the long bones. Histological examination of bone shows arrested osteogenesis with marked attenuation of trabeculæ On the other hand when fractures are sustained there is abundant formation of osteoid not only in surrounding callus but also within the bone fragments (Fig 633) It has been claimed by Albright that the union of fractures is accelerated by administration of testosterone propionate and methyl testosterone[1] Such treatment should also be used in borderline cases Simpson has indicated the many intermediate grades between patients who show the fully developed Cushing syndrome and so-called "normal" women who are past middle age, have some degree of excess adiposity and hypertrichosis, and in whom fractures may unite more rapidly with the aid of suitable hormonal therapy[2]

Pathological fractures from cortisone treatment—Since it is known that hormones of the adrenal cortex are katabolic, or at least anti-anabolic, and that in promoting breakdown of protein to glucose they suppress osteogenetic activity—thus causing the osteoporosis of Cushing's syndrome—it is not surprising that cortisone treatment of rheumatoid arthritis and other diseases should predispose to pathological fracture. Experimental administration of cortisone-acetate in rabbits has been shown to retard all phases of the healing of fractures[3, 4]

Hypopituitarism (Frohlich's syndrome)—The syndrome of hypopituitarism described by Frohlich in 1901 is a deficiency disease of the anterior pituitary. In the first case he described there was a "cranio-pharyngioma" with delayed sexual development and adiposity from spread of the tumour to the hypothalamus These patients are dwarfed, or at least below average height, and defective secretion of testicular and adrenal androgens causes retarded growth, slenderness of bones, weakness of muscles, and laxity of ligaments There is often separation and displacement of the upper femoral epiphysis, usually in patients who are excessively fat and much above normal weight.

Hypogonadism (adipose gynandrism)—The classical Frohlich syndrome from pituitary tumour is very rare indeed, but on the other hand hypogonadism with delayed sexual maturity and excessive adiposity without a detectable pituitary lesion occurs commonly—it has been described by Simpson[5] as *adipose gynandrism* The patients are of average or more than average height, extreme adiposity is associated with red or purple lineæ distensæ on the trunk (Fig 634) Delayed sexual maturity, the female type of pelvic girdle, and absence of pubic and axillary hair are often associated with spontaneous displacement of the upper femoral epiphyses during adolescence The displacement may occur so gradually that it is not recognised The patient often complains of no more than an occasional limp There may sometimes be pain but it is often referred to the knee, and between attacks of pain and limping the boy seems to be normal apart from his fatness Nevertheless careful examination shows limitation of internal rotation movement of the hip joints Routine antero-posterior radiographs may show no conclusive evidence of displacement, but if lateral radiographs

[1] Albright, F. *Recent Progr Hormone Res*, 1947, **1**, 293
[2] Simpson S L *J Bone Joint Surg* 1950 **32-B**, 734
[3] Ragan, C, *et al* *Proc Soc Exper Biol Med*, 1949, **72**, 718
[4] Ingle D J *J Endocrin*, 1950, **10**, 1312
[5] Simpson, S L *Brit med J*, 1950, **1**, 692, *J Bone Joint Surg*, 1950, **32-B**, 738

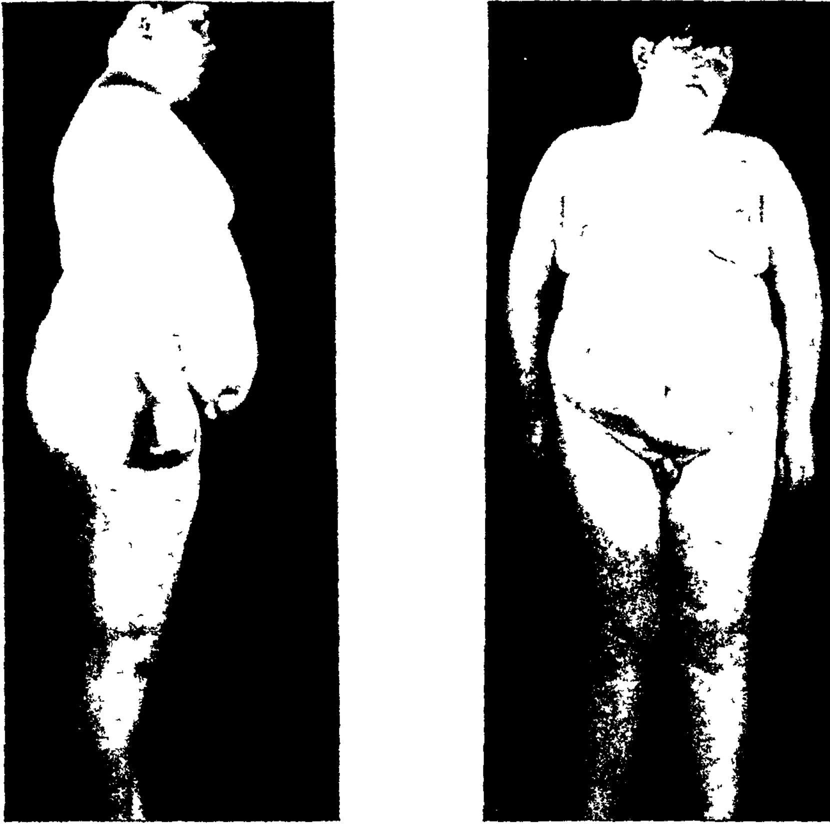


FIG. 634

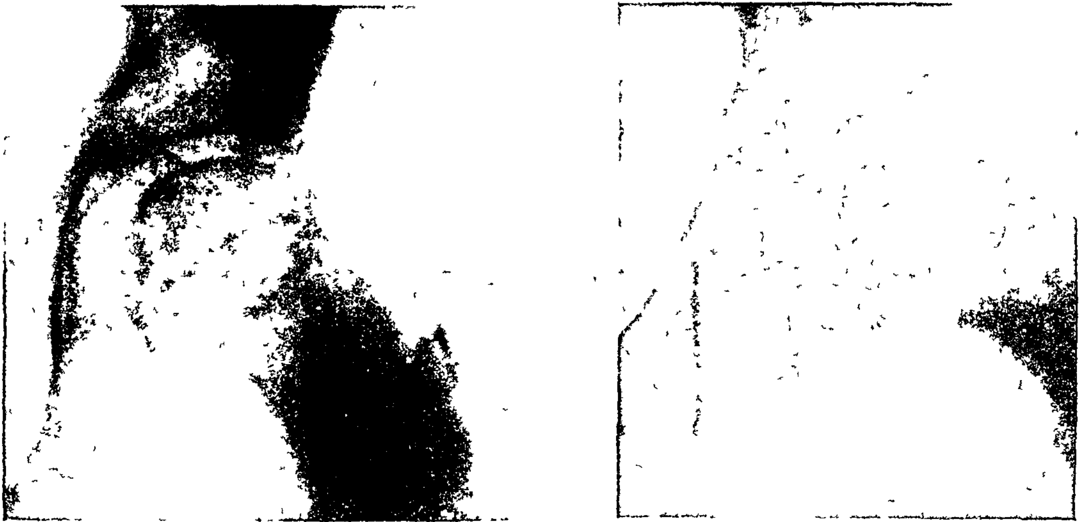


FIG 635

Adiposе gynandrism

(Frohlich type of hypopituitarism)

This boy grew fat at the age of four years (Fig. 634) He ate a lot but was always hungry At fourteen years he measured 5 feet 6 inches and weighed 213 pounds (over 15 stones) Genitalia small; no pubic or axillary hair, testes undescended Glucose tolerance curve normal, 17-K and Gona excretion low, blood urea 22 mgm, serum-albumen 4 8, serum-globulin 3 0; serum-cholesterol 270 mgm. For three months he had limped, and radiographs show early slipping of the upper femoral epiphysis, more obvious in the lateral projection (Fig 635) Treated by bed rest, and thyrotrophic and gonadotrophic hormones (*Presented at Staff Conference, Robert Jones and Agnes Hunt Orthopadic Hospital, by Mr Lloyd Griffiths, investigations at Manchester Royal Infirmary*)

are taken in the Lauenstein position, separation and backward displacement of the upper femoral epiphysis is more obvious (Fig. 635). It is sometimes possible to prevent increasing displacement by simple bed rest, and to treat the constitutional deficiency by thyrotrophic and gonadotrophic hormones, but very often the tendency to displacement of the epiphysis is so pronounced that fixation must be secured by the insertion of a three-flanged nail from below the trochanter (Figs. 636-637)

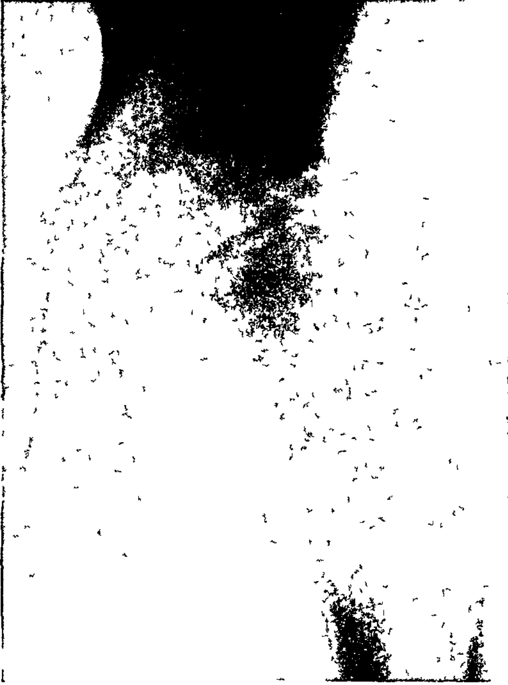




FIG 636 FIG. 637

Adipose gynandrism (Frohlich type of hypopituitarism)

In another patient, similar to the one shown in Figures 634-635, displacement of the upper femoral epiphysis increased very rapidly Within two weeks, even while he was in hospital, there was change from the position shown in Figure 636 to that in Figure 637. The displacement was reduced by very gentle manipulation and a three-flanged nail was inserted. (*London Hospital case*)

Lawrence–Moon–Biedl syndrome—This syndrome, which is often familial, is characterised by hypogonadism with general adiposity, mental retardation, retinitis pigmentosa and polydactyly [1] The association of fatness with infantilism suggests hypopituitarism, but the underlying pathological disorder has not yet been established.

DISUSE AND SENILE OSTEOPOROSIS OF BONE

Disuse atrophy of bone was at one time attributed to decalcification or halisteresis It was thought that demineralisation left the cellular structure of bone intact, but histological study shows that the trabeculæ themselves are destroyed There is failure of normal osteoblastic apposition while normal osteoclastic resorption continues This may be seen in the fracture of the patella shown in Figure 136, page 68, in which the proximal fragment lost its blood supply so that for a time there was complete arrest of vital activity with cessation of both apposition and resorption of bone, the

[1] Lawrence, J Z, and Moon, R C "Four Cases of Retinitis Pigmentosa occurring in the same Family and accompanied by General Imperfection of Development" *Ophthal Rev*, 1866, 2, 32.

dead trabeculæ remaining as a control index of former thickness ; meanwhile the living distal fragment with its free blood supply showed disuse atrophy with reduction in thickness of all bone trabeculæ and complete disappearance of some. The greater the functional inactivity, the more complete is the failure of bone apposition in keeping pace with osteoclastic resorption, and the more severe is the osteoporosis.

In every fracture, no matter how treated, there is at first osteoporosis. This can be minimised by prompt immobilisation of the fragments and continued active contraction of the muscles , but if the fragments are not immobilised adequately and functional disuse is aggravated because muscle contraction is painful, or if despite adequate immobilisation no attempt is made to institute active exercises, then the osteoporosis is increased. It may continue even after union of the fracture because inactivity causes adhesion of muscles and stiffness of joints, with still greater difficulty in practising exercise. The vicious circle of functional inactivity causing disuse change, and disuse change inhibiting functional activity, is completed in Sudeck's acute post-traumatic atrophy which was discussed on pages 53-55.

Disuse osteoporosis causing fracture—Osteoporosis induced by a fracture may be so severe as to cause secondary pathological fracture One patient, a young and vigorous soldier at the peak of fitness, sustained a fracture of the femur which was treated elsewhere by open reduction and plating The wound became infected and, before the fracture was united, two long miserable years had been spent in the removal of sequestra and the draining of many abscesses. By then there was such osteoporosis that in early attempts to walk he sustained an almost spontaneous fracture of the neck of one femur, and a central fracture-dislocation of the other hip. Several months later, in an attempt to mobilise the stiff knee, the femur was refractured at the supracondylar level. The bone was as soft as a rotten apple

Poliomyelitis causing fracture—Disuse osteoporosis may also arise when there is extensive paralysis of muscle In the management of patients with acute poliomyelitis, care must always be taken to avoid fracture of long bones from careless handling of a limb. Fractures often occur in the shaft of the femur.

Senile osteoporosis—In the same way that osteoporosis arises from functional inactivity because apposition of new bone fails to keep pace with normal osteoclastic resorption, it develops in old age because there is general retardation of cellular proliferation. Replacement of outlived osteocytes demands resorption of surrounding bone, and this goes on while osteoblastic apposition of new bone fails Thus the bone trabeculæ become progressively thinner, the fat-content increases, and the cortex is thinned. This general osteoporosis predisposes to trochanteric fractures of the femur, especially in elderly women, and sometimes to fractures of the shafts of long bones. The softening of bone and the predisposition to fracture are most pronounced in the spine. There is bi-concavity of vertebral bodies with ballooning of intervertebral discs because porotic bone has much less resistance to weight-bearing stress than fibro-cartilaginous discs. Increasing reduction in the depth of vertebral bodies, particularly in the dorsal region, causes diminution of stature and kyphosis, often with aching pain which is aggravated by degenerative arthritis of intervertebral and interlaminar

joints. From time to time this vague discomfort and general aching may be accentuated by acute attacks of agonising pain, almost immobilising in its intensity, and it is then found that one or more vertebral bodies have been crushed Pathological crush fractures are sustained from trivial incidents such as stooping to lift a weight, using the starting-handle of a car, or even stumbling on a carpet Radiographic examination shows complete crushing of one vertebral body, often with evidence of former crushing of other vertebræ in the lumbar and dorsal regions

Differential diagnosis—Porosis of the spine with bi-concavity of vertebræ, disappearance of bone trabeculation, and susceptibility to pathological fracture may arise also in osteomalacia, leukæmia, diffuse myelomatosis, hyperparathyroidism and Cushing's syndrome. Care should be taken not to conclude that because a patient is between sixty and eighty years of age osteoporosis is necessarily senile in type (see Figs. 678-680). Investigation of blood cells, blood chemistry and urine is essential. In osteomalacia the serum-phosphorus is low, in leukæmia there is an abnormal blood count; in diffuse myelomatosis the serum globulin is raised and there may be proteoses in the urine; in hyperparathyroidism serum-calcium is high and serum-phosphorus low; and in Cushing's syndrome there is albuminuria, hypercholesteræmia and hyperglycæmia In uncomplicated senile osteoporosis the blood cells are normal, the serum-calcium and serum-phosphorus are at normal levels, and alkaline-phosphatase is normal unless there has been a recent fracture, when it is raised In nearly half the cases there is achlorhydria, the significance of which is not yet understood.

Treatment of fractures in senile osteoporosis—No constitutional treatment is known by which to stimulate bone formation in senile osteoporosis. It may be that the atrophy of old age represents underfunction of the steroid-producing glands—but we do not know. The intake of calcium and phosphorus should certainly be increased by giving calcium phosphate, 3 grammes daily A sufficient supply of vitamin D should be assured by giving from 5,000 to 10,000 international units If there is achlorhydria, dilute hydrochloric acid should be given with meals. Hormonal treatment is discussed on the next page, but the management of senile osteoporosis is essentially symptomatic This does not mean that there must be despair. Fractures always unite and pain can always be relieved—and the very worst treatment is to let the patient think that she is "too old" In explaining softening of bone and susceptibility to fracture it should be said that the bones "are not as young as they were"—a subtle distinction from saying that they are too old, and one which keeps the patient happy, and it must be emphasised that the fracture will unite quickly and that function will be restored The fracture will indeed unite quickly, and function may well become normal for a patient of that age

It is quite unnecessary to reduce acute crush fractures of the spine by hyperextension, and it is seldom necessary to apply a plaster jacket. The patient should be rested in bed for about two weeks on a firm mattress with a soft pillow at the mid-dorsal level. After the first few days hyperextension exercises should be encouraged Meanwhile a posterior spinal support of the type used by Hugh Owen Thomas and Robert Jones should be ordered—but it must be of the lightest metal, and it must be slender, fitted with a comfortable abdominal corset and with well-padded shoulder straps, and it

should be moulded closely to the existing curves of the spine Such a support will be needed indefinitely in order to relieve aching pain and prevent increasing deformity, and furthermore to protect from spontaneous fractures In the beginning, great care should be taken to meet every whim of the dear lady who must wear it—because otherwise she will not wear it at all Occasionally the pain of a crush fracture in senile osteoporosis is so severe that it is not relieved by bed rest, and the patient, old as he or she may be, values the protection of a plaster jacket This should be applied while the patient sits or stands—not in the prone "two-table" position—and without any attempt to change the curves of the spine After six or eight weeks the plaster jacket should be replaced by a carefully moulded posterior spinal support

Menopausal osteoporosis—The greater frequency of senile osteoporosis in women than in men and the frequent onset of symptoms at about the age of fifty suggest that menopausal deficiency of gonadal hormones may be an important factor It is worth trying intramuscular administration of testosterone propionate 50 mgm on alternate days for the first week and then methyl testosterone 20 mgm , and diethylstilbœstrol 1 mgm., daily by mouth Very often the pain is relieved within a few weeks and there is improvement in general well-being, although radiographic evidence of increased ossification of bone is seen only after many months and sometimes not at all

DYSTROPHIC CYSTS AND FIBROUS DYSPLASIAS OF BONE

Fibrocystic disease or osteitis fibrosa cystica was at one time believed to represent a clinical entity characterised by fibrous replacement of bone with cyst formation and many osteoclastic cells It is now recognised to be no more than a manifestation of many different diseases von Recklinghausen's osteitis fibrosa cystica has already been differentiated and is better described quite simply as hyperparathyroidism . osteoclastoma or giant-cell tumour of bone has also been differentiated . and various types of solitary bone cyst, hæmorrhagic cyst, non-osteogenic fibroma; and monostotic or polyostotic fibrous dysplasia are being identified

Solitary cyst of bone (dystrophic cyst)—One of the commonest causes of pathological fracture in young adolescents is a solitary dystrophic cyst in the upper humerus, femur or tibia, and sometimes in other long bones It begins in childhood or adolescence as a metaphyseal lesion with destruction of bone, slight expansion, and thinning of the cortex. There is no periosteal reaction or new bone formation such as occur when there is infection. The differential diagnosis from osteoclastoma is sometimes difficult and may necessitate biopsy, but as a rule the differentiation is quite clear Dystrophic cysts are seen in children between the ages of five and fifteen, whereas osteoclastomas are rare before the age of twenty (though they may occur before that age, despite all that has been said to the contrary) , cysts develop in the metaphyseal region and tend to occupy the shaft, whereas osteoclastomas, having started in the metaphysis, quickly invade the epiphyseal plate if it is still present and extend to the articular cartilage; cysts are more common in males, and osteoclastomas in females , cysts occur in the upper ends of the humerus, femur and tibia, whereas osteoclastomas are more common in the lower femur, upper tibia and lower radius

The first evidence of a dystrophic cyst is often a fracture from trivial

injury. If a fracture is sustained it always unites quickly and may even promote healing of the dystrophy, the cyst being obliterated by newly formed callus ; there should certainly be no haste in considering surgical intervention (Figs. 638-639) The fracture should be treated on conventional lines by immobilisation in splints or plaster, but if the cyst fails to heal it should be explored and grafted in order to prevent recurrent fracture. Part of the attenuated cortex should be removed , the brown or clear fluid within the cyst should be evacuated and the thin layer of tissue lining the wall curetted , the space should then be filled with cancellous chips of bone cut from the ilium Healing is usually complete, but some cysts are more aggressive and may persist despite bone grafting Radiotherapy is then advisable but special



Fig. 638



Fig 639

Dystrophic cyst with pathological fracture

Dystrophic cyst of the upper end of the tibia in a boy aged twelve years who sustained a fracture through the cyst from trivial injury The limb was immobilised in plaster after correcting the forward angulation The fracture united within three months and the cyst underwent spontaneous obliteration Operation was unnecessary , the dystrophy was cured by the fracture

care must be taken to use low dosage—not more than 2,000 rontgen and often less—in order to avoid injury to the epiphyseal plate and premature fusion

Hæmorrhagic cyst or benign bone aneurism—When a solitary cyst is explored, it is sometimes found to be full of blood which flows freely from the cavity but without pulsation (Fig 640) Lack of thrombosis indicates that there has been a free, though perhaps sluggish, circulation. Histological examination of connective tissue in the lining wall shows many engorged and dilated vascular channels The lesion was described by Ewing as a benign bone aneurism [1] and by Lichtenstein as an aneurismal bone cyst [2]—both titles being unfortunate because the cyst has nothing whatever to do with aneurisms The clinical and radiographic signs are similar in nearly

[1] Ewing, J "Neoplastic Diseases A Treatise on Tumors" Philadelphia and London W B Saunders & Co 4th ed , 1940, 323

[2] Lichtenstein, L "Aneurismal Bone Cyst" *Cancer*, 1950, 3, 279

all respects to those of simple dystrophic cysts except that hæmorrhagic cysts tend to lie more in the shaft than the metaphysis, and they may develop in the vertebræ or in flat bones such as the scapula Pathological fractures from trivial injury are often sustained, but they unite without difficulty If the cyst is curetted and filled with bone chips it usually heals

Non-osteogenic fibroma of bone—This localised fibrous dysplasia also occurs in the ends of long bones in children and adolescents and causes expansion and thinning of the cortex, often with pathological fracture.[1] The clinical and radiographic appearances are similar to those of a simple dystrophic



FIG 640

Hæmorrhagic cyst of the ulna

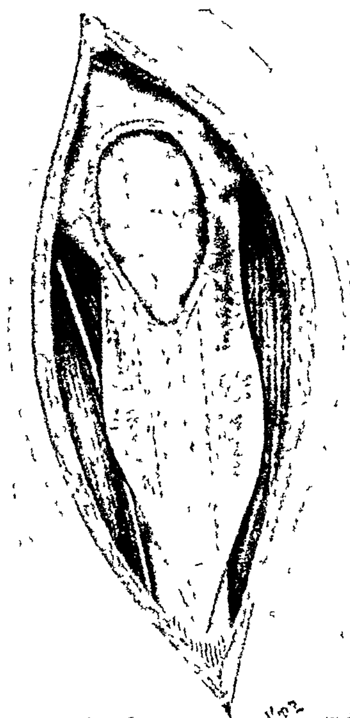


FIG 641

Non-osteogenic fibroma of the humerus

Figure 640 shows a hæmorrhagic cyst, or benign aneurism of the ulna, in a girl aged nine years The cyst was curetted and filled with chips from the bone bank Figure 641 shows a non-osteogenic fibroma of the upper end of the humerus with pathological fracture The ridge of bone at the site of the united fracture can be seen on each side of the window cut in the thin cortex to expose the fibroma which was removed—a whole-thickness graft being slid up from the shaft to fill the defect.

cyst, but when exposed at operation the area is found to be filled with a solid mass of yellow or brown fibrous tissue (Fig 641) Among the masses of dense fibrous tissue there may be giant cells, and also foam cells containing lipoid, thus accounting for the view that the dystrophy represents a burnt-out lipoid granuloma More probably non-osteogenic fibroma of bone, or "metaphyseal fibrous defect," as it is sometimes called, arises from developmental failure of metaphyseal ossification, the distance of the lesion from the epiphyseal line depending on the particular stage of growth at which the failure was most pronounced

Monostotic and polyostotic fibrous dysplasia—Fibrous dysplasia may occur more diffusely in a bone (monostotic dysplasia) or in several bones of

[1] Jaffe, H , and Lichtenstein L "Non-osteogenic Fibroma of Bone" *Amer. J Path* , 1942, 18, 205



FIG. 642



FIG. 643

Fracture complicated by fibrous dysplasia

A corporal in the Royal Air Force sustained a fracture of the shaft of the femur from severe injury. It was not a spontaneous or pathological fracture, but radiographic examination showed fibrous dysplasia of the femur and tibia, and union of the fracture was slow. After long immobilisation it united soundly.

one or more limbs (polyostotic dysplasia) with involvement not only of long bones but also of the pelvis and skull. There is often a curiously unilateral distribution. This, too, is believed by Jaffe to arise from a congenital developmental failure of ossification,[1,2] but the fact is that we do not know the etiology. The essential pathological change is replacement of large areas of bone by masses of dense fibrous tissue arranged in whorls, with small islands of fibre-bone produced by membrane ossification but with no tendency to multiplication of osteoclasts or to cyst formation. There may be lipoid-laden histiocytes or xanthoma cells which have suggested a relationship with lipoid granulomatosis.[3]

The association of predominantly unilateral fibrosis of bone with pigmentation of skin and precocious puberty is known as Albright's syndrome.[4] Pigmentation from increased deposition of melanin in the deeper layers of the epidermis occurring in one or more sites of variable size, has been noted in about one-third of reported cases; precocious puberty has been observed less often. In girls with multiple bone lesions there may be early enlargement of the breasts and premature growth of pubic and axillary hair; menstruation has been known to begin within a few months of birth. Premature puberty with development of secondary sex characters during the first decade has been reported also in boys.[5] Occasionally there has been hyperthyroidism. It seems almost certain that polyostotic fibrous dysplasia and certainly Albright's syndrome, must have a hormonal basis, but we do not know what it is.

Pathological fractures and severe deformity may occur in childhood, but sometimes there is no evidence of abnormality until adult life, and even then it may be disclosed incidentally; there is, indeed, an almost infinite gradation in the severity of the dysplasia as it occurs in different patients. A corporal in the Royal Air Force engaged in athletic and gymnastic activities, and only when he sustained a fracture of the femur from considerable violence was fibrous dysplasia recognised, the fibrosis of bone accounting for slow union of the fracture (Figs. 642-643); but another patient suffered his first fracture at the age of four, was bedridden for nearly twenty years, has now sustained scores of fractures, and only last week fractured his ulna when he turned over in bed (Figs. 650-652). Severe cases usually become apparent in infancy by reason of the general thickening of several bones, gross deformity, and susceptibility to fracture. The blood chemistry is normal, thus differentiating this type of dysplasia from vitamin-resistant rickets and hyperparathyroidism; and radiographic examination distinguishes it clearly from dyschondroplasia. There is diffuse thickening with loss of distinction between cortex and medulla so that the whole bone appears homogeneous, but sometimes with areas of such complete fibrous replacement as to give the appearance of a cyst (Fig. 644). There is no longitudinal metaphyseal striation, or fan-like appearance of the pelvis, such as occurs in dyschondroplasia.

Even when the first evidence of the dysplasia arises in childhood it may be relatively benign. A boy aged fourteen sustained a fracture of the

[1] Lichtenstein, L., and Jaffe, H. L. "Polyostotic Fibrous Dysplasia." *Arch. Surg.*, 1938, **36**, 874.
[2] Lichtenstein, L., and Jaffe, H. L. "Fibrous Dysplasia of Bone." *Arch. Path.*, 1942, **33**, 777.
[3] Snapper, I. "Medical Clinics on Bone Diseases." New York: Interscience Publishers, 2nd ed., 1949.
[4] Albright, F., Butler, A. M., Hampton, A. O., and Smith, P. "Syndrome characterised by Osteitis Fibrosa Disseminata." *New Eng. J. Med.*, 1937, **216**, 727.
[5] Warrick, C. K. "Polyostotic Fibrous Dysplasia—Albright's Syndrome." *J. Bone Joint Surg.*, 1949, **31-B**, 175.



Fig. 644



Fig. 645



Fig. 646

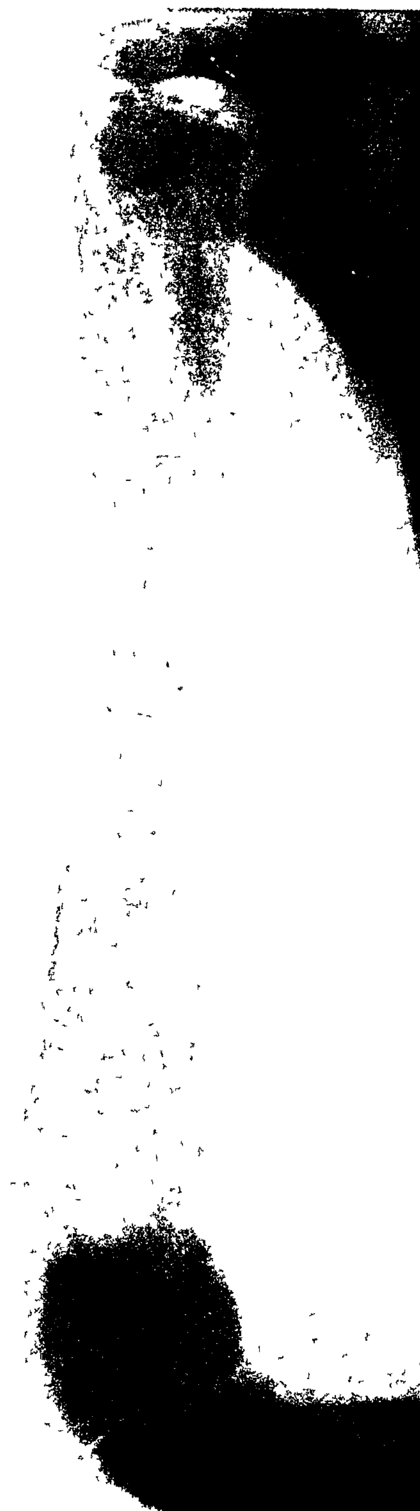


Fig. 647

Polyostotic fibrous dysplasia

A boy aged fourteen years sustained a fracture of the lower shaft of the humerus from trivial injury. Radiographic examination showed that the fracture was through an area of fibrous replacement of bone, and there was also diffuse thickening of the humerus, radius and metacarpal bones on the radial side (Figs. 644-646). The fracture was treated conservatively and it united soundly; moreover, the fibrous area ossified (Fig. 647).

humerus from trivial injury, and radiographic examination showed that it was a pathological fracture through an area of fibrous replacement. The humerus, the radius and several metacarpal bones on the radial side showed the characteristic signs of polyostotic fibrous dysplasia (Figs 644-647). Nevertheless the fracture united after simple conservative treatment and the fibrous defect healed by new osteogenesis.



Fig 648



Fig 649

Monostotic fibrous dysplasia

A male aged forty-three years sustained a fracture through a cystic area in the tibia from trivial injury. The cyst was curetted and bone grafts were implanted It appeared that the lesion had healed, but two years later another spontaneous fracture was sustained (Fig. 648). The blood chemistry was normal The area of bone showing fibrous dysplasia was excised, the gap being filled with cancellous grafts cut from the ilium, stability was maintained by onlaid cadaveric grafts of whole-thickness bone (Fig 649). Histological examination confirmed the diagnosis.

Another patient sustained a fracture of the mid-shaft of the tibia, and it was found that the injury had been sustained through an area of fibrous replacement. The region was curetted and filled with a bone graft. Healing appeared to be complete, but two years later when the patient sustained another spontaneous fracture at the same site there was still evidence of active fibrous replacement (Fig. 648) The whole thickness of the shaft was excised, including the periosteum and the subperiosteal layers, and it was replaced by cancellous chips cut from the ilium, stability being maintained by onlay grafts of boiled cadaveric bone (Fig. 649)

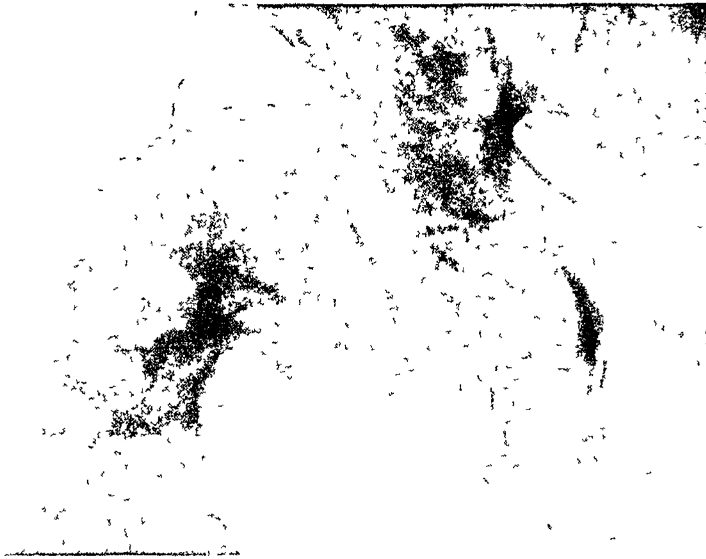


Fig 650



Fig 651



Fig. 652

Polyostotic fibrous dysplasia with many pathological fractures and very severe deformities

This patient with polyostotic fibrous dysplasia sustained his first fracture at the age of four. Since then he has sustained scores of fractures—some treated rather stupidly with bits of wire. He was bedridden for nearly twenty years After corrective osteotomies the deformities were corrected enough for him to stand and walk, and all the osteotomies united quickly But he still needs surgical appliances to prevent recurrent deformity

It must be reiterated that we are still unaware of the etiology of polyostotic fibrous dysplasia and still unable to cure it Sometimes no more is needed than to prolong the period of immobilisation of a fracture, and sometimes we can help with bone-grafting operations: in very severe cases surgical appliances are needed to protect from deformity Not until we know the hormonal basis—if there is one—shall we be able to treat it rationally.

Leontiasis ossea—One of the finest specimens of a skull with leonine contours from tremendous masses of supra-orbital, malar and maxillary bone was dissected without permission from the face and scalp of a cadaver. and replaced by a plaster cast without anyone knowing it had been done, it was an amazing feat of skilful dissection The extraordinary hyperostotic thickening of leontiasis ossea is almost certainly a variant of polyostotic fibrous dysplasia, and not of Paget's osteitis deformans as was often believed

PAGET'S DISEASE OF BONE—OSTEITIS DEFORMANS

This is another disease of unknown etiology, and the misfortune of not knowing the underlying cause is still greater than in polyostotic fibrous dysplasia because Paget's disease[1] occurs so much more frequently. It is



FIG 653
Multiple incomplete cracks of the subcutaneous surface of the tibia in Paget's disease



FIG 654
Sarcoma arising at the site of a pathological fracture of the femur in Paget's disease

perhaps one of the commonest diseases of bone The only satisfaction we have is that progression is very slow indeed Although it begins before the age of forty, or even thirty, symptoms seldom arise before the sixth or seventh decades—but it causes pathological fracture, and in a proportion of cases there is sarcomatous transformation (Figs 653-654)

[1] Paget, Sir James "Osteitis deformans" *Medico-Chir Trans*, 1877, 60, 37

The disease is often localised to one bone, the tibia being involved more often than other long bones. For ten or twenty years the patient is unaware of abnormality; there may be clinical evidence of thickening of the shaft, reduced sharpness of the crest, or local heat from increased circulation, but there is little or no pain. As years go by the thickening becomes more obvious and there may be deep aching pain, sometimes with a tendency to forward and outward bowing of the shaft of the bone. If the disorder extends as far as the articular surface of the knee joint secondary degenerative arthritis may develop, with stiffness and pain especially on first standing after sitting for long periods.



FIG. 655

One of the commonest pathological fractures in osteitis deformans is a strictly vertical fracture through the femoral neck. Special care should be taken in nailing this fracture because the bone is liable to split, but careful nailing has proved successful.

Radiographic examination shows an irregular deposit of many trabeculæ of new bone, first on the subperiosteal surface and then on the medullary surface, so that the distinction between cortex and medulla is lost (Figs. 580–591). Osteoid or woven bone is laid down, and during active stages of the disease no attempt is made to convert this young bone to mature lamellated bone. Unbridled formation of immature osteoid is reflected in the high level of serum-alkaline-phosphatase. In relation to the area of bone involved, the phosphatase level is higher than in any other disease. In all other respects the blood chemistry is normal—there is no change in the serum levels of calcium, phosphorus or protein, and there is no abnormality in urinary excretion.

Although clinical manifestations may be confined to one bone—the tibia, femur or clavicle, one metacarpal or phalanx, or sometimes a single



Fig 656



Fig 657

Osteitis deformans—Paget's disease

Pathological fracture of the shaft of the tibia in Paget's osteitis deformans, un-united only because it was not immobilised adequately (Fig 656). There is abundant formation of osteoid in this disease and fractures nearly always unite soundly if they are immobilised properly On the other hand, fractures of the neck of the femur may be complicated by avascular necrosis of the femoral head which makes union more difficult (Fig 657) Nevertheless this fracture united after simple immobilisation in plaster for five months



[illegible]

Osteitis deformans

[illegible]

vertebra—there is usually radiographic evidence of thickening and irregular trabeculation of the pelvis Symptoms seldom arise from osteitis deformans of the innominate bones unless there is extension to the hip joints and pain from secondary arthritis, but radiographic evidence confirms the diagnosis in doubtful cases Similarly there may be involvement of the skull with thickening of the calvarium, but again without symptoms Even the symptom that formerly was classical—the inability of a man to wear his old hat—has disappeared with the passing of the fashion of bowlers and silk-hats Quite a different type of change in the skull has been recognised recently as a feature of Paget's disease Instead of uniform thickening with blurred contour from generalised apposition of osteoid, there may be wide areas of porosis of the calvarium with no change in adjacent bones (Fig 590)

To the surgeon who treats fractures the particular significance of Paget's disease is that replacement of mature lamellated bone by immature woven bone causes fragility despite thickening Lack of normal resistance to weight-bearing is shown first in a tendency to bowing of the shafts, and then in multiple incomplete cracks through the thickened cortex on the convex side of the bowed bone (Figs 653 and 658) Any of these cracks may be converted into complete fractures from trivial injury In the management of such fractures two important principles should be observed 1) there must be adequate and sustained immobilisation exactly as in the treatment of any other fracture—the fragments will certainly unite by abundant formation of osteoid or woven bone and repair is usually rapid, the only cause of non-union, other than sarcomatous transformation, being inadequate immobilisation (Fig 656), 2) pre-existing deformity should usually be corrected at the site of fracture, because if bowing persists there will still be predisposition to pathological fracture Even when there has been no fracture it is sometimes wise to correct deformity by osteotomy, and advantage should certainly be taken of any fracture that is sustained

Fractures of the shaft of the tibia should be immobilised in plaster after alignment has been restored Fractures of the femoral shaft are best treated by traction in a Thomas' splint It may be that some fractures of the shaft of the femur in Paget's disease lend themselves to immobilisation by an intramedullary nail, but this technique should be considered only with caution because encroachment of new bone on the medullary canal may cause seizing of the nail when it is half-way in, or even disastrous splitting of the bone Similarly, fractures of the femoral neck, which are very common in Paget's disease and occur in a strictly vertical plane unlike that of ordinary fractures in this situation (Fig 655), may best be treated by the intramedullary fixation of a three-flanged nail, but special care must be taken to avoid splitting the bone One disastrous early experience in which I split a femoral neck as if it was a piece of rotten wood made me advise against nailing in earlier editions of this book, but there is no doubt that the technique has merit, and that it may be safe and successful if slender nails are driven cautiously through the bone The fracture of the neck of the femur in a patient with Paget's disease, shown in Figure 657, was complicated by vascular disturbance of the femoral head—but it united after conservative treatment alone, as it might not have done if the bone had been normal. In this disease there is vigorous formation of osteoid and, although final consolidation may be slow, primary union is rapid.

PRIMARY AND SECONDARY TUMOURS OF BONE CAUSING PATHOLOGICAL FRACTURE

Pathological fractures are often caused by primary or secondary tumours of bone, particularly tumours of the osteolytic type For example, multiple myeloma is a destructive neoplasm occurring in many sites without reparative bone formation, and fractures are sustained in more than 60 per cent of cases, whereas Ewing's endothelioma with its medullary destruction is characterised also by subperiosteal apposition of new bone in many "onion-layers," and only 5 per cent of these tumours are complicated by fracture The incidence of fracture in various bone tumours was estimated by Geschickter and Copeland[1] as multiple myeloma, 62 per cent., cysts and cystic tumours, 45 per cent, secondary carcinoma, 15 per cent., giant-cell tumour, 14 per cent, osteosarcoma, 15 per cent, Ewing's tumour, 5 per cent.

Classification of bone tumours—A simple classification of bone tumours based on that of the American Bone Sarcoma Registry in 1939 will suffice for our purpose

Benign tumours

Chondroma	Fibroma
Hæmangioma	Giant-cell tumour

Primary malignant tumours

Osteosarcoma	Ewing's tumour
Chondrosarcoma	Myeloma or plasmocytoma
Fibrosarcoma	Malignant giant-cell tumour

Metastatic bone tumours

Carcinoma of the breast, thyroid and prostate glands

Hypernephroma	Epithelioma

Chondroma, osteochondroma, myxochondroma—Chondromas of the phalanges or metacarpals are often disclosed for the first time by pathological fracture (Fig 659) These tumours usually develop during the first two decades of life, and the commonest sites are the short bones of the hands or feet. At first there is painless expansion near the end of the bone, sometimes extending throughout the shaft Radiographs show no trabeculation as in osteoclastoma, or periostitis and general porosis as in syphilitic or tuberculous dactylitis The well-defined cystic area is demarcated from normal bone and surrounded by an intact but thin cortex Crack fractures may be sustained from trivial injury, and indeed this is one of the most frequent causes of pathological fracture because chondromas of the phalanges are common tumours and about 40 per cent of them are complicated by fracture

Treatment of fracture through a chondroma of a phalanx—The finger should be immobilised for two or three weeks by means of a finger splint or light plaster slab applied to the flexor surface of the slightly flexed digit. Some weeks later, when joint movement has been restored, the tumour should be excised and grafted in order to prevent recurrent fracture. The bone is exposed on its lateral aspect between the flexor and extensor tendons,

[1] Geschickter, C F, and Copeland M M "Tumors of Bone" *Amer J Cancer* New York, 1936

but with care to avoid damage to the digital nerves; part of the cortex is removed After curetting, the cavity is filled with cancellous chips of bone from the ilium, or perhaps still better with a block of cancellous bone and thin cortex cut accurately to shape from the upper end of the ulna of the same limb (Figs 660-661). Fractures through chondromas of the phalanges always unite, and the tumours respond well to surgical treatment but not to irradiation.

Massive chondromas of long bones and bones of the pelvis—Large chondromas sometimes develop in the metaphyseal region of long bones, or in the pelvis or other bones of the trunk Multiple chondromas in dyschondroplasia and diaphyseal aclasia were discussed on pages 380-383. Solitary tumours, no less massive, may arise even when there is no developmental disease, and they often continue to increase in size long after cessation of epiphyseal growth. In the pelvis they may compress the bladder or cause ureteric or intestinal obstruction. These massive tumours are potentially malignant but, even after biopsy, it is often difficult to distinguish benign chondromas



Fig. 659



Fig. 660



Fig. 661



Fig. 662

Tumours and cysts of the phalanges

Figure 659 shows a chondroma with crack fracture. Figures 660-661 show a similar tumour treated by curetting and grafting Figure 662 shows an implantation dermoid with fracture

of vigorous growth from malignant chondrosarcomas that will metastasise. The distinction is sometimes more academic than practical because even proved chondrosarcomas are not radio-sensitive, and their growth cannot be controlled by X-ray treatment Massive chondromas should therefore be treated by local excision—or if that is quite impossible by amputation of the limb Chondroma of the pelvis with visceral obstruction must usually be treated by hindquarter amputation.

Hæmangioma of bone—Hæmangioma usually occurs in the vertebræ or skull of young adults. There may be honeycombed rarefaction of one vertebra with collapse and wedging of the body which must be distinguished from the crushing of a simple compression fracture Hæmangiomas also arise in the short bones of the hand and foot with characteristic soap-bubble appearance (Figs 663 and 665). As a rule the bone is destroyed so completely that it bends rather than fractures. The tumours respond well to irradiation, and surgical excision with bone grafting is seldom indicated.

Fibroma of bone—Rare benign tumours of bone include fibromas and lipomas. Fibrous transformation of bone in monostotic and polyostotic fibrous dysplasia and non-ossifying fibroma of bone were discussed on page 403.



Fig 663



Fig. 664

Angioma of bone

An angioma of the fifth metacarpal bone was treated by radiotherapy The symptoms were relieved and the bone reossified (*London Hospital case*)



Fig 665

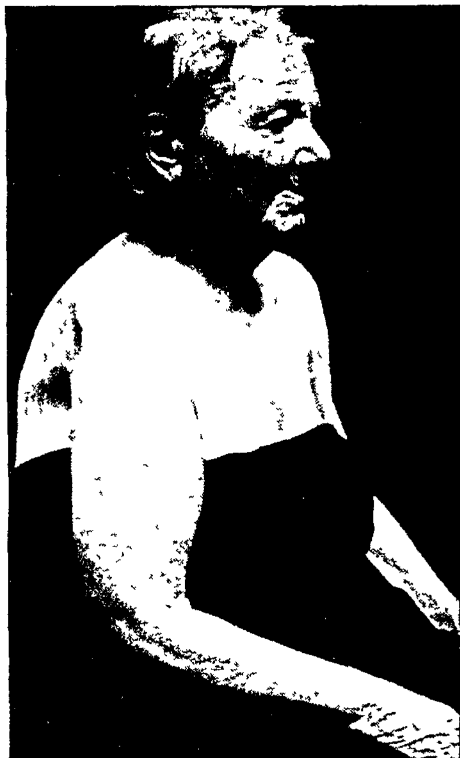


Fig 666

Angioma of bone

Angioma of the shaft of the humerus showing characteristic soap-bubble appearance. There was a pathological fracture, but the old lady lived happily to a ripe age.

Giant-cell tumour of bone—There is still disagreement about the pathology of giant-cell tumours of bone, and confusion is increased by the variability of aggression that may occur in tumours of apparently similar histological type. On the one hand there seems to be a link between simple dystrophic cysts with giant cells in their lining wall and solid giant-cell tumours with multinucleated cells distributed evenly through a stroma of spindle cells: and on the other hand it is difficult to distinguish between giant-cell tumours that are essentially benign and tumours that recur locally after excision or even cause metastasis. Thus a "giant-cell tumour process" has been postulated with every stage of benignity, aggression and malignity from simple dystrophic cysts at one end of the scale to frankly malignant tumours at the other.[1] The difficulties are increased by the fact that histological examination of a giant-cell tumour may give no suspicion of maligant propensities, and yet secondary tumours arise in distant tissues—sometimes with a cellular structure that appears no less benign than that of the primary tumour.[2] In other cases, whether from the beginning or after excessive irradiation, the cellular structure of both primary and secondary tumours is obviously malignant (Figs 668-670).



FIG. 667

Giant-cell tumour

Giant-cell tumour of the upper end of the fibula with pathological fracture treated by excision of the diseased bone.

Dystrophic fibrous cysts were discussed on page 401 They occur more often in boys than in girls, and in the metaphyseal region of long bones with a tendency to extend to the shaft; injury causing fracture often results in spontaneous healing of the cyst Giant-cell tumours seldom occur before the age of twenty (but there is no strict dividing line of age, and true giant-cell tumours may occur in adolescents and sometimes in infants). This tumour is more common in girls. It begins in the metaphysis, but there is rapid spread to the articular surface, the epiphyseal plate being invaded if it has not already closed. Even articular cartilage is less resistant to invasion from this tumour than from most others, and benign tumours may spread across the joint and invade the next bone.[3]

The bone is expanded, at first asymmetrically, the common sites being the upper end of the tibia, upper end of the fibula, lower end of the femur, and lower end of the radius. It may also arise in the head of the femur, pelvis, sacrum or spine, and occasionally in other bones There is no subperiosteal new bone formation such as occurs in Brodie's abscess and many osteosarcomas. Radiographs show trabeculation throughout the

[1] Platt, H "Osteoclastoma or Giant-cell Tumour of Bone" *J. Bone Joint Surg*, 1949, **31-B**, 157
[2] Russell, D "Malignant Osteoclastoma" *J Bone Joint Surg*, 1949, **31-B**, 281
[3] Windeyer, B W, and Woodyatt, P B "Osteoclastoma" *J Bone Joint Surg*, 1949, **31-B**, 252



Fig. 668



Fig 669



Fig. 670

Malignant osteoclastoma

Occasionally a tumour which at first appears to be a simple osteoclastoma betrays malignant tendencies and, despite surgical treatment or radiotherapy, progresses to complete destruction of the bone (*London Hospital case treated by Mr Frank Ellis*)

tumour with thinning and sometimes perforation of the cortex; there is usually a line of condensation separating the tumour from the normal bone of the shaft. At operation the tumour is bright red in colour and very vascular, hæmorrhage sometimes causing extensive blood cysts within the tumour mass. Less aggressive tumours may be more firm, and grey or even white in colour.

Complete excision of giant-cell tumours is perhaps the treatment of choice when it can be done without causing functional incapacity—but this applies only to the fibula (Fig 667). The lower end of the radius has been excised and replaced by a transplant of the upper end of the fibula, or by a graft from the tibia implanted into the carpal bones, and the upper end of the tibia has been excised and the knee joint fused but these operations are justified only when there is extensive invasion of articular cartilage. In other cases better results can be achieved by more conservative treatment. It must be emphasised, moreover, that even after complete excision of the tumour, islands of osteoclastomatous tissue may remain in the soft tissues and continue to grow, invading the bone that has been transplanted.

I believe that giant-cell tumours are best treated by radiotherapy The diagnosis should first be confirmed by open biopsy (not punch biopsy, which is often misleading). The dosage of irradiation must be low—not more than 2,000 rontgen in adults, and about 1,000 rontgen in younger patients. Higher dosage not only damages the epiphyseal plate (which may already be injured by the tumour) but may cause malignant transformation in previously benign tumours

Others believe that it is better to expose the tumour, curette the cavity, swab the walls with phenol, and fill the space with cancellous iliac chips, irradiation being reserved for tumours that recur after surgical treatment But there is strong evidence to suggest that the aggression of a giant-cell tumour is increased by the injury of surgical intervention and particularly by the osteoclastic activity that is needed to resorb and replace bone grafts Moreover it is agreed fairly generally that the results of combined surgical intervention and irradiation are less satisfactory than either alone.

Fractures should therefore be treated by immobilisation in splints or plaster—a Thomas' splint with skin traction being used for tumours of the upper end of the tibia and lower end of the femur and a plaster slab for tumours of the lower end of the radius As soon as convenient the diagnosis should be confirmed by open biopsy through a short incision. Irradiation should then be arranged, immobilisation being continued until the fracture has united and until healing of the tumour is sufficient to prevent crushing and deformity.

Osteosarcoma is the commonest malignant tumour of bone, two-thirds of the cases occurring between the ages of ten and thirty years More than half are in males The usual sites are the lower end of the femur, upper end of the tibia, and upper end of the humerus, less commonly it arises in the scapula, ilium, fibula and forearm bones The first symptom is pain, which often precedes the appearance of a tumour. In tumours of low malignancy there may have been slight pain for many months before advice is sought, but in highly malignant tumours there is severe pain with quick appearance of a tumour which grows rapidly and soon causes emaciation and death. In

nearly all osteosarcomas the pain is persistent and constant, without the intermissions that characterise the pain of bone infections

The clinical and radiographic features and the incidence of pathological fracture vary according to the bone-producing or bone-destroying qualities of the tumour Sclerosing types of osteosarcoma may be stony-hard tumours of slow growth There is radiographic evidence not only of medullary and cortical destruction but also of periosteal new bone formation with "sun-ray spicules" of ossification at right angles to the shaft and "Codman's reactive triangle" where bone is laid down beneath the raised periosteum at the margin of the tumour In these cases pathological fracture is rare. On the other hand osteolytic and telangiectatic osteosarcomas are soft and may almost appear to fluctuate; there may be pulsation and a bruit, and pathological fracture is not uncommon.

In general, osteogenic sarcomas are not radio-sensitive and the treatment that offers greatest hope of survival is immediate amputation. It is nearly always advisable to confirm the diagnosis by open biopsy. The possible risk of disseminating the tumour by open biopsy is far less important than the risk of amputating a limb for low-grade osteomyelitis or some other lesion that resembles sarcoma Even with amputation the five-year survival rate is low, and the modern tendency is to irradiate the tumour first, using dosage so high that the limb is virtually destroyed by the irradiation—a dosage that would be impossible unless the decision to amputate had already been taken

Chondrosarcoma occurs usually between the ages of thirty and fifty in the long bones, pelvis and ribs The tumours may arise at the site of pre-existing chondroma (p 415). Growth is usually slow and the prognosis is more favourable than in many other sarcomas of bone. Chondrosarcoma is not radio-sensitive and should be treated by amputation or wide resection

Fibrosarcoma is a comparatively rare tumour, occurring usually in the femur It is composed of spindle cells which have no bone-forming properties, often arising from the periosteum and developing slowly as a firm tumour outside the shaft of the bone Even when it develops in the medulla it is very slowly destructive, and pathological fractures seldom occur The tumour is not radio-sensitive but the prognosis is relatively favourable and wide local excision of parosteal growths is often justifiable. If the tumour recurs, or if it arises primarily in the medulla, the limb should be amputated

Ewing's tumour occurs early in life, usually between the ages of ten and twenty years, in the shafts of long bones and very occasionally in the flat bones The clinical course is often slow Pain is at first intermittent, and there are spontaneous remissions of growth with febrile attacks which are sometimes accompanied by leucocytosis, and radiographic evidence of medullary and cortical destruction accompanied by considerable new bone formation (Fig 671) The differentiation from chronic osteomyelitis is difficult and is sometimes established only after biopsy, or after observing the striking initial response to irradiation (Fig 672) Although irradiation of Ewing's tumour of bone nearly always relieves pain, arrests periosteal new bone formation, and causes shrinking of the tumour, recurrence is inevitable and the patient dies within two or three years Irradiation, followed soon after by amputation, is the treatment of choice.



Fig. 671



Fig. 672



Fig. 673

Ewing's tumour

diffuse osteolytic lesion of the upper femoral shaft was associated with periosteal reaction causing " ers" The diagnosis of Ewing's sarcoma was made after biopsy. The immediate response to irrad s excellent (Fig 672) but, as invariably happens, the tumour recurred (Fig 673) The patient died two years and the diagnosis was confirmed at autopsy. (*London Hospital case*)

Myeloma (multiple myelomatosis, plasma-cell tumour, plasmocytoma) is one of the most rare malignant tumours of bone It arises from plasma cells of the marrow, usually in the second half of life from the ages of forty to seventy years. A still smaller group of tumours is derived from myelocytes, erythroblasts or lymphocytes. The tumours are almost invariably multiple and they show a curious predilection for the bones of the trunk—the spine, ribs, sternum, and then in order of frequency the skull, femora, pelvis and clavicle Multiple myelomas of bone are sometimes associated with extra-



Fig 674



Fig 675

Multiple myelomatosis

Pathological fractures of the shafts of humerus and femur from multiple myelomatosis There were Bence-Jones proteoses in the urine, the serum globulin was raised (see Figs 676-677)

medullary tumours in the nasopharynx; and occasionally myelomas arise in the larynx, air sinuses or upper air passages without bone involvement Of all malignant tumours of bone it is the one with the highest incidence of pathological fracture. Crush fractures of vertebræ, spontaneous fractures of ribs, or fractures of the shafts of one or both femora occur in at least two-thirds of patients with this disease (Figs 674-677)

Pain is the first manifestation and it is usually in the back In so far as the patients are usually elderly, and pain in the back from senile osteoporosis or intervertebral arthritis is to be expected at this age, the diagnosis is sometimes overlooked The radiographic evidence of multiple

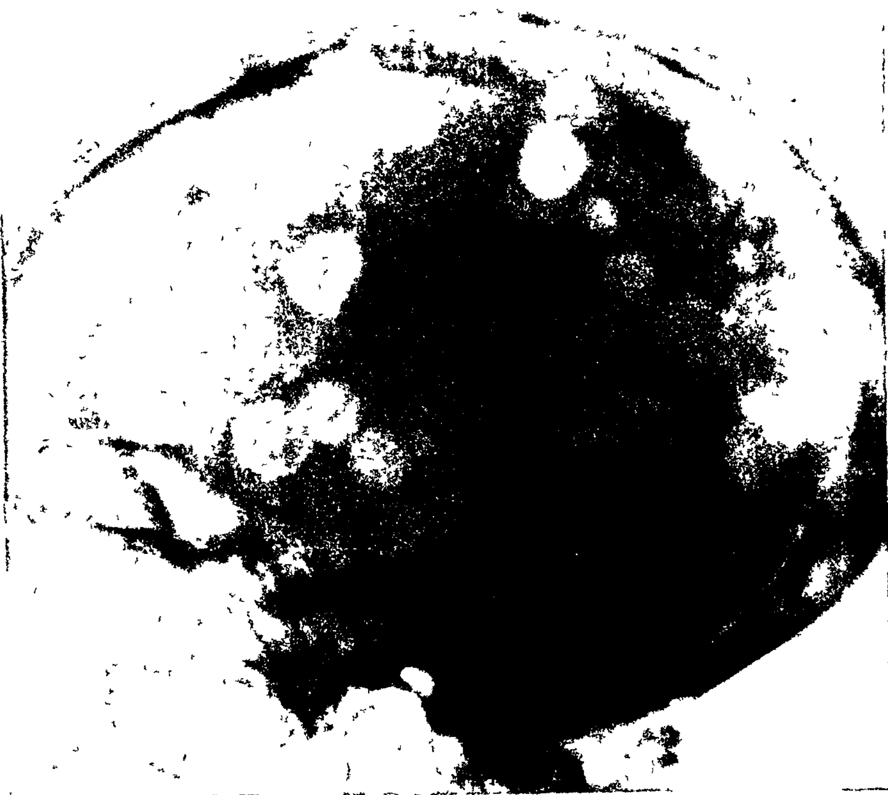


Fig. 676



Fig 677

Multiple myelomatosis

Same case as that shown in Figures 674-675 The patient died Post-mortem radiographic examination of the skull shows multiple tumours with typical punched-out defects In the frontal region there is a shadow of the tumour associated with one of the bone defects (Fig 677)

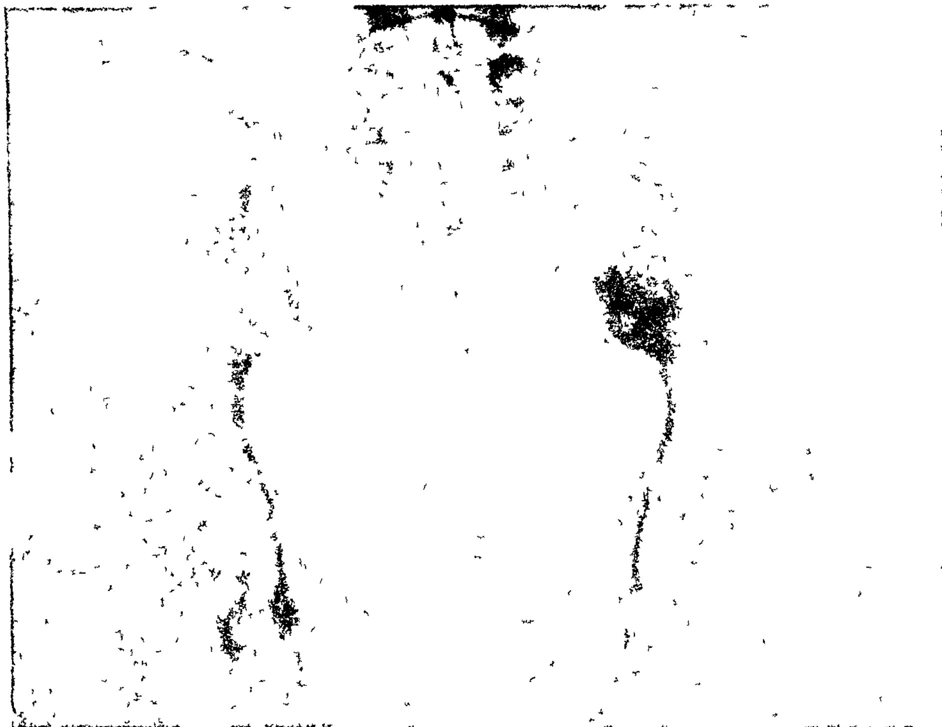


Fig 678

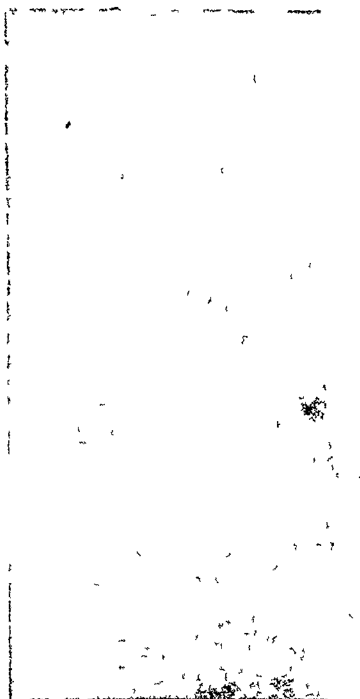


Fig 679



Fig 680

Myelomatosis simulating senile osteoporosis

A man aged seventy-eight years sustained compression fractures of the spine simply from lifting weights Radiographs showed general porosis of the pelvis and spine with cupping of vertebral bodies resembling senile osteoporosis (Fig 679) But the plasma globulin was found to be greatly raised (9 9 gm per cent, the normal being not more than 2 gm per cent) There was depletion of plasma albumen from the normal 4 to 2 1 gm per cent Serum calcium was raised to 12 6 mgm per cent There were no Bence-Jones proteoses in the urine, but radiographs of the humerus and other long bones suggested that in addition to general porosis there were multiple small cysts (Fig 680) Sternal marrow puncture was arranged (see Fig 681)

cystic tumours, most of them 1 or 2 cm. in diameter occurring not only in the vertebræ but also in the skull and other bones, may make the diagnosis fairly clear, but in other cases the tumours are more confluent and there is generalised destruction of all vertebral bodies (Figs 678-680) In these cases confirmation of the diagnosis depends on analysis of the urine, estimation of serum proteins, and punch biopsy of the sternal marrow.

Urinalysis—In 1848 Bence-Jones described an unusual protein in the urine of patients suffering from multiple myelomatosis, a proteose that is precipitated below 60° C disappears on boiling, and reappears on cooling. But Bence-Jones proteosuria is found only in 65 per cent. of patients with

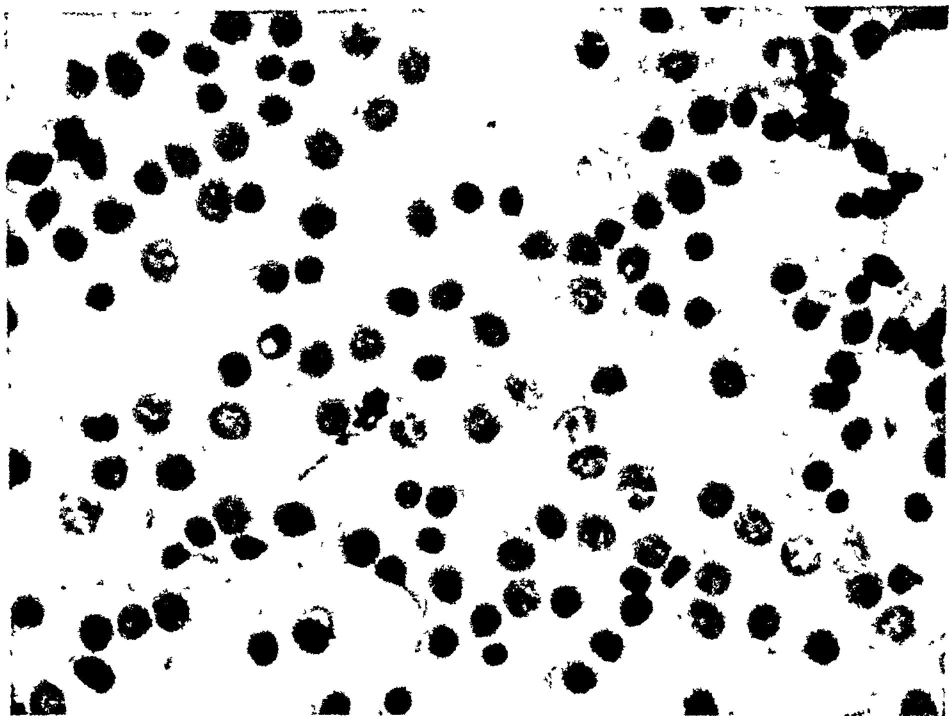


Fig. 681

Myelomatosis

Same case as that of Figures 678-680. Sternal marrow puncture showed typical plasma myeloma cells (*Investigations by Dr Godwin and Dr Robertson Department of Clinical Investigation, The London Clinic, microphotograph* × 470, *by Mr John King, Bernhard Baron Laboratories, London Hospital*)

myeloma of bone (and there is no record of it occurring at all in extramedullary myelomas) It cannot therefore be relied upon as a diagnostic test.

Estimation of blood proteins—On the other hand serum protein is always raised—the protein being of the same chemical composition as Bence-Jones protein in the urine. On taking blood for chemical analysis it may be found difficult to obtain serum by reason of the high content of fibrin. If oxalated blood is examined it is at once evident that there is marked increase in plasma globulin with depletion of albumen The normal levels are · fibrinogen 0·2, albumen 4·0, and globulin not more than 2 0 grammes per cent In the patient whose radiographs are shown in Figures 678-680 (at first thought to be a simple example of senile osteoporosis with crush fractures) the fibrinogen was raised to 0·39 grammes, the serum albumen was lowered to 2·1 grammes

and the serum globulin was raised to 7·3 grammes per cent The total serum protein may even be as high as 18·4 grammes per cent High plasma-globulin causes a secondary rise in serum-calcium, even up to 13 mgm per cent, and also of serum inorganic phosphate up to 6 or 7 mgm per cent, thus distinguishing the blood chemistry from that of hyperparathyroidism Blocking of renal tubules with Bence-Jones proteins may cause raised blood urea and even acute uræmia

Sternal marrow puncture—If the diagnosis is still in doubt after urinalysis and estimation of serum proteins, punch biopsy of sternal marrow and histological examination of the marrow smear is advisable (Fig 681)

Treatment—Multiple myelomatosis is a fatal disease and most patients die within a few months or at the most within a few years When a myeloma appears to be solitary the expectation of life may be longer, especially if the tumour is irradiated The tumours are radio-sensitive and after treatment there is usually regression and reossification If the disease is already widespread, involving the spine, pelvis, femora and skull, the discomforts of irradiation should be avoided, no more can be done than to make the patient comfortable until he dies, but with localised involvement, irradiation can be expected to relieve pain Fractures of the spine should not be reduced, but a light posterior spinal support may be fitted Fractures of the femoral shafts are usually best treated in a Thomas' splint—but in occasional cases intramedullary nailing may give greater comfort during the few months or years of survival

Metastatic tumours of bone—Skeletal metastases occur in the later stages of cancer of the breast, kidney, adrenal, prostate, thyroid and lung. As a rule generalised bone metastases represent the terminal stages of visceral carcinoma, but sometimes a solitary metastatic tumour is found even before the first symptom or clinical sign of the primary tumour. Indeed, Geschickter and Maseritz found no clinical evidence of a primary growth in one-sixth of the metastatic bone tumours they studied [1] The differential diagnosis of these solitary metastases presents considerable difficulty because they may resemble primary growths so closely The age incidence is usually much higher than in primary tumours—it is from fifty to seventy years, and the bones most often involved by spread through the blood stream are those with the highest proportion of marrow—the vertebræ, pelvis, upper end of the femur, upper end of the humerus, and the skull and ribs Pathological fractures often arise from trivial injury, especially in tumours of osteolytic type from primary carcinomas of the breast, kidney and thyroid

Carcinoma of the breast gives rise to metastases in the spine, pelvis, femur, skull, ribs and humerus in the order given As a rule the lesions are multiple and they are usually osteolytic The deposit is first in the medulla and there is then secondary invasion of the cortex, sometimes with diffuse mottling within the area of destruction, but with no periosteal reaction (Fig 682). The lesions are less clearly punched out than the typical deposits of diffuse myelomatosis are In the spine, destruction of a vertebral body but without collapse—indicating that destruction has been accompanied by replacement with tumour tissue—serves to differentiate metastatic deposits from tuberculosis and other inflammatory diseases (Fig 684) When fractures are sustained they should be immobilised by splints or plaster because this is

[1] Geschickter, C F, and Maseritz, I H *J Bone Joint Surg*, 1939, 21, 314

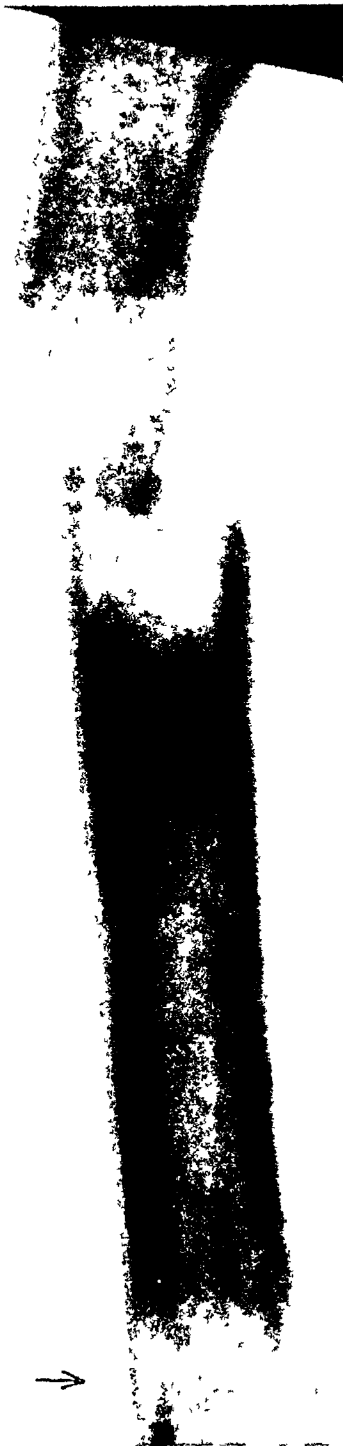


FIG. 682



FIG. 683



FIG. 684

Secondary carcinoma of bone

Figure 682 shows a pathological fracture through a metastasis in the upper shaft of the femur from carcinoma of the breast. There is a second deposit in the lower shaft. The pathological fracture in Figure 683 is from a secondary hypernephroma in the humerus associated with another deposit in the scapula. Figure 684 shows the typical appearance of secondary carcinoma of the spine, despite extensive bone destruction there is minimal collapse because the bone is replaced by tumour tissue

the best way of relieving pain—the fractures always unite and good function may be restored In fractures of the neck of the femur through the site of a metastatic deposit there should be no hesitation in introducing a three-flanged nail The patient whose radiographs are shown in Figures 685-686 was riddled in every bone with carcinomatosis . she was emaciated and semi-conscious from the drugs given to relieve her pain and she had not sat on a bed-pan from the day the fracture was sustained because the pain was so



FIG. 685



FIG 686

Carcinomatosis with fracture of the neck of the femur

Pathological fractures of the femoral neck from secondary malignant disease should usually be treated by the methods of internal fixation used for simple fractures This patient, with deposits in every bone from a neglected carcinoma of the breast, sustained a pathological fracture of the neck of the femur and was unable to move, even to sit on a bed-pan, by reason of pain in the hip After the fracture was nailed she was comfortable and happy Similarly, intramedullary nailing of pathological fractures of the shafts of long bones often gives comfort and freedom from pain

great Was ever a prospect more gloomy ? Yet after nailing of the fracture all pain disappeared She began to sit up and move in her bed, and with treatment by massive doses of vitamin B-12 she is now a happy and cheerful woman. her fracture united Similarly in fractures of the shaft of the femur from metastatic deposits the insertion of an intramedullary nail gives immediate comfort and relieves the patient from the added discomforts of splints and plaster casts

Carcinoma of the thyroid may cause bone metastasis with pathological fracture. The primary tumour is often small and is sometimes disclosed only at post-mortem examination.

Carcinoma of the prostate is usually associated with very slowly growing metastases in the lumbar vertebral bodies and pelvis. The invasive powers of the secondary growth are more than equalled by the new bone formation induced, and the characteristic radiographic feature of bone metastases from prostatic carcinoma is that of dense sclerosis (Fig 687). The ability of this tumour to stimulate osteoblastic bone formation seems to arise from the high content of acid-phosphatase in prostatic cells and the cells of

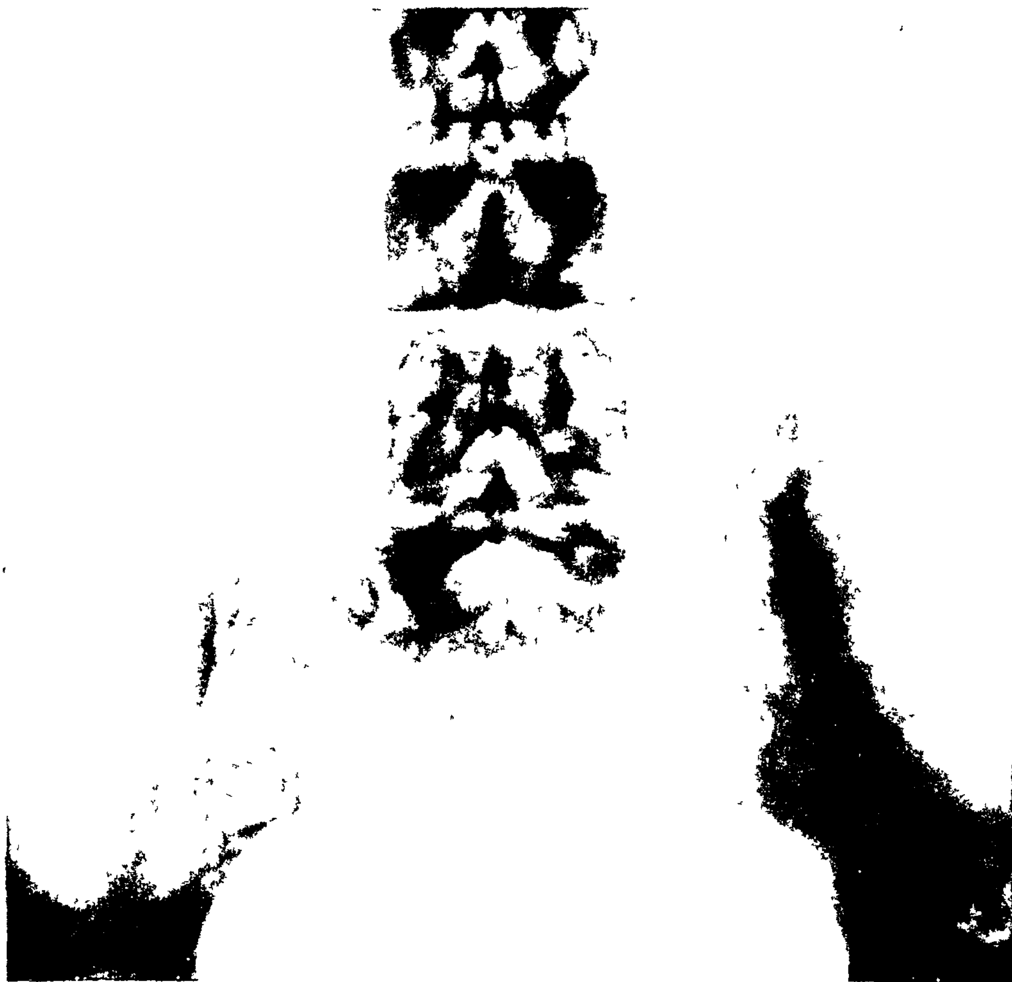


FIG. 687

Secondaries from carcinoma of prostate

There is abnormal density of the third lumbar vertebral body and of the left ilium near the sacro-iliac joint arising from secondary deposits from carcinoma of the prostate. The serum acid-phosphatase was raised.

secondary neoplasms from the prostate. It may be reflected in a high level of serum acid-phosphatase. So much new bone is formed that pathological fractures are rare, and if they are sustained they unite rapidly The dramatic, but usually temporary, response of the metastases of prostatic carcinoma to œstrogen-therapy and castration is still being studied.

Hypernephroma (renal carcinoma)—The metastases of hypernephroma have a predilection for bone The lesion is usually destructive and pathological fractures are common (Fig 683). Very often the metastasis occurs as a solitary bone deposit and ten-year cures have been reported



FIG. 688

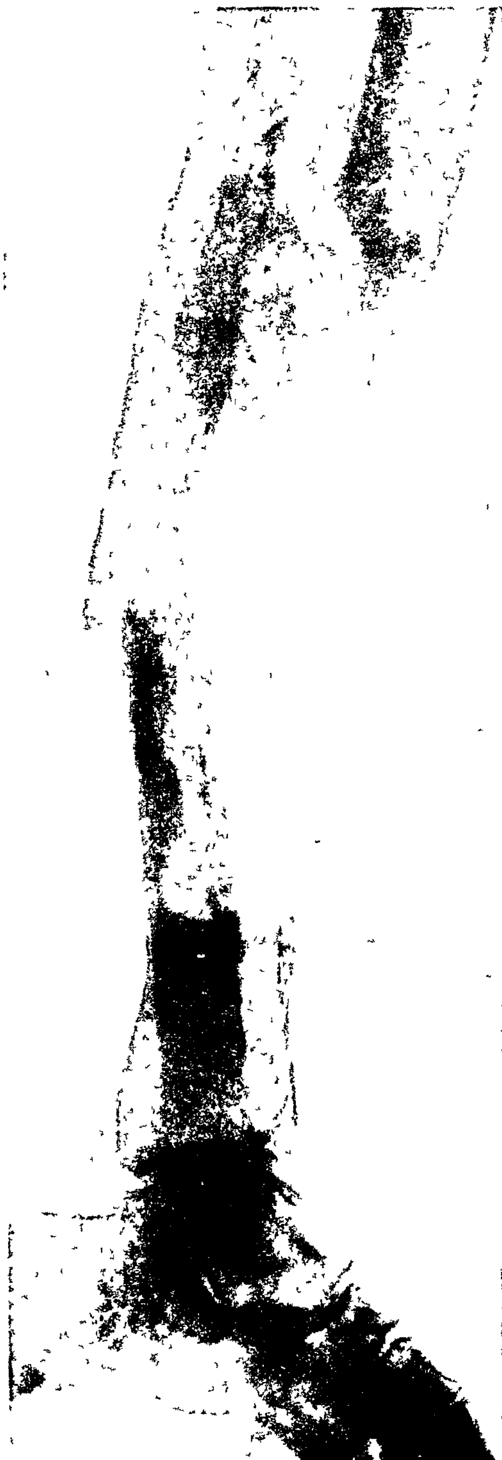


FIG. 689

Epithelioma of bone

An epithelioma of the skin occurring at the site of chronic varicose ulceration invaded the tibia and eventually destroyed a very large part of it, the fibula also sustaining fracture (Fig 689). *(Robert Jones and Agnes Hunt Orthopaedic Hospital case.)* Pathological fracture of the tibia with similarly extensive destruction is often seen after tropical ulceration with malignant transformation

after excision of the involved bone.[1] On the other hand metastases in bone have sometimes first appeared many years after removal of the kidney—the interval being as long as ten years in some reported cases [2]

Epitheliomatous invasion of bone—Direct invasion of the mandible or maxilla from epithelioma of the lip or tongue is well known—but similar invasion may occur in the shafts of long bones and cause pathological fracture. An example is illustrated in Figures 688-689. A patient suffered varicose ulceration of the leg for many years and eventually an epithelioma developed in the margin of the ulcer. The shafts of the tibia and fibula already showed diffuse subperiosteal new bone formation from the œdema and hyperæmia of periosteum associated with such long proximity to a region of active infection (Fig 688), but with continued growth of the epithelioma there was rapid and extensive invasion of bone, first causing a pathological fracture of the tibia, then extensive destruction of most of the shaft of the bone, and finally a fracture of the fibula (Fig 689). The limb was amputated. Similarly extensive destruction of bone from direct epitheliomatous invasion is seen after tropical ulceration of the shin complicated by malignant change in the ulcer.

DISEASES OF THE MARROW CONSTITUENTS

Tumours of the marrow including plasma-cell tumours or myelomas, and the rare tumours that may arise from myelocytes, erythroblasts or lymphocytes, were discussed on pages 422-426. There is also a group of blood and marrow diseases including granulomatoses of bone, the pathology of which is uncertain although it is clear that bone is replaced by lipoid tissue and that pathological fractures often occur.

Lipoid granulomatosis—Hand–Schuller–Christian disease—In this disease bone is replaced by granulomas made up of lipoid-containing reticulo-endothelial cells, the cells often being yellow by reason of their content of cholesterol esters and described as xanthoma cells. The deposits are usually small, varying from less than 1 cm. to about 2 cm in diameter, but sometimes there is general confluence of many deposits causing widespread areas of destruction, especially in the skull. In the case shown in Figures 690-692 the disease had given rise to no symptoms, but radiographic investigation after injury to the hip joint showed several small cystic areas in the pelvis Similar areas of bone destruction were then found in the skull and the upper end of the humerus The blood cholesterol was normal but biopsy disclosed many reticular cells expanded by deposition of cholesterol.

Gaucher's splenomegaly—In Gaucher's disease, which occurs especially in the Hebraic races, the reticulum cells are infiltrated with kerasin. The deposits are more widespread, especially throughout the lower shafts of the femora which are ballooned, and the upper ends of the tibiæ, and the spleen and liver are greatly enlarged by similar deposits. Pathological fractures through the weakened bone are very common. The patient whose radiographs are shown in Figures 693-696 had sustained many fractures of the right femoral shaft and finally the limb was amputated, on the assumption that the fractures were a consequence of uncontrollable osteomyelitis. There were, however, typical changes in the opposite femur, pigmentation of

[1] Albrecht *Arch Klin Chir*, 1905, 77, 1073.
[2] Broster, L R "Secondary Hypernephroma Femur with Spontaneous Fracture." *Brit J Surg*, 1923, 11, 287.

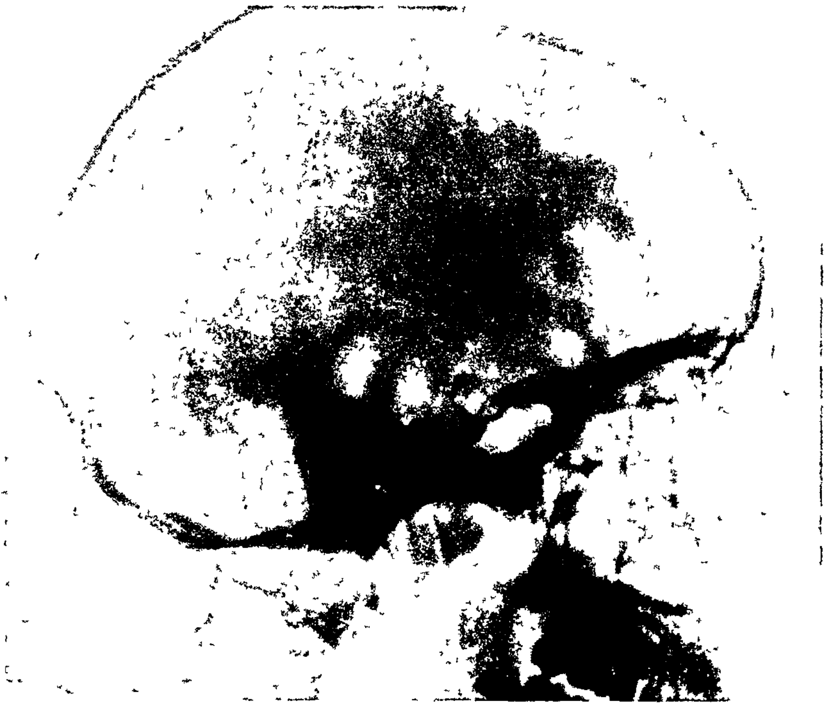


Fig. 690



Fig 691 Fig 692

Lipoid granulomatosis of bone—Hand–Schuller–Christian disease

A boy sustained an injury to the hip joint and radiographs showed small cystic areas in the femora and pelvis Further radiographs showed similar granulomatous cysts in the neck of the humerus and skull The diagnosis of Hand–Schuller–Christian lipoidosis was confirmed by punch biopsy

(London Hospital case, radiography by Dr Jupe)

skin, and lipoid deposits in the conjunctiva. Extensive lipoid deposit in the upper shaft and neck of the femur often interferes with the blood supply of the femoral head, and avascular necrosis with crushing of dead bone is one of the most typical features of Gaucher's disease of bone. One patient with avascular necrosis of the femoral head from kerasin-lipoid deposits in the femoral neck developed severe flexion adduction deformity of the hip; she was seriously incapacitated and quite unable to walk (Figs. 697-698) An osteotomy was performed at the subtrochanteric level; it was through





Fig. 693 Fig 694

Lipoid granulomatosis of bone—Gaucher's disease

(For legend see Figures 695-696 on the next page.)

an area of bone heavily infiltrated with lipoid deposits and the osteotomy was performed with some trepidation, but with the knowledge that in this disease pathological fractures usually unite Consolidation of the osteotomy was slow and after four months' immobilisation in plaster, when the possibility of non-union was feared, cancellous chips cut from the ilium were impacted round the site of osteotomy After six months, union was soundly consolidated and this girl has led a happy life ever since (Fig 699). Realignment of weight-bearing permitted regeneration of the femoral head

and relieved the symptoms that would have persisted from secondary arthritis Six years have now elapsed and she still has normal function in the limbs—though there is persistent abdominal discomfort because the liver is so enlarged that it is half-way down to the umbilicus, the spleen having been removed ten years ago

Eosinophilic granuloma of bone—In this disease many areas of bone are replaced by granulation tissue with collections of large phagocytic macro-



Fig 695

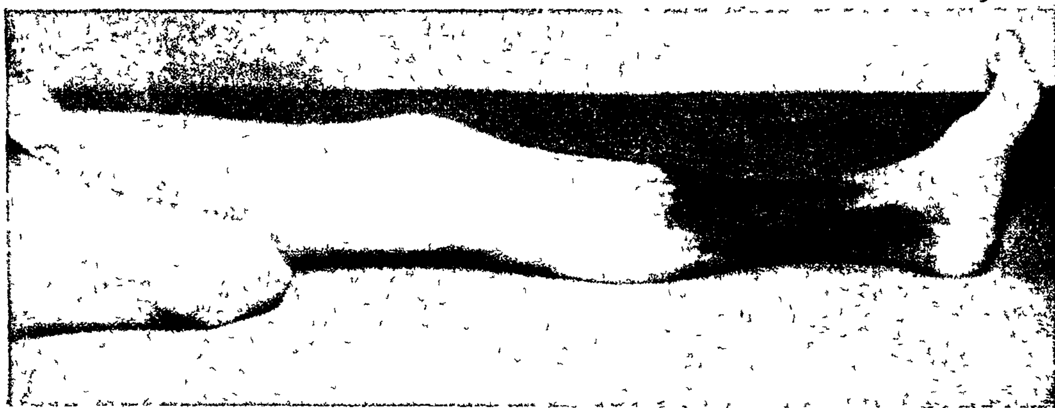


Fig 696

Lipoid granulomatosis of bone—Gaucher's disease

This Israeli boy sustained so many spontaneous fractures of the shaft of the right femur that the limb was amputated (before we saw him), the condition had been diagnosed as "chronic osteomyelitis" There were typical changes of lipoid granulomatosis of Gaucher's type (Fig 693) with expansion of the opposite femoral shaft and avascular necrosis of the femoral head (Fig 694), pigmentation of skin (Fig 696) and lipoid deposits in the conjunctiva (Fig 695)

phages, including reticulo-endothelial cells, and eosinophil cells There are sometimes areas of hæmorrhage and necrosis, and in later stages there is replacement by fibrous tissue The common sites are the skull, vertebræ, ribs, pelvis and long bones The disease usually occurs in children or adolescents and it causes local tenderness without obvious swelling or constitutional disturbance, pathological fracture is often the first manifestation The blood cytology and blood chemistry are normal, except

for occasional eosinophilia and sometimes a raised alkaline phosphatase. The diagnosis is established by histological examination of fragments removed at biopsy. Simple curetting heals any one focus and the granulomas are radio-sensitive, but in any event healing tends to occur spontaneously within twelve months or two years



Fig. 697



Fig. 698



Fig. 699

Gaucher's disease with avascular necrosis of the femoral head

A frequent complication of Gaucher's disease of bone is avascular necrosis of the femoral head from lipoid deposits in the neck which interfere with the blood supply In this Jewish girl there was recurrent flexion-adduction deformity and severe disability from pain, stiffness, apparent shortening and limp. A displacement osteotomy was therefore performed, even although necessarily through diseased bone Union of the osteotomy was slow, and at the third month it was supplemented by cancellous bone chips cut from the ilium. After six months' immobilisation, consolidation of the osteotomy was sound. There has been no further disability in five years except for a passing attack of pain and limping from a new lipoid deposit in the ischiopubic ramus

Letterer-Siwe's disease (reticulo-endotheliosis or non-lipoid histiocytosis) is a condition of hyperplasia of macrophages without lipoid storage affecting infants below the age of two years, characterised by enlargement of the liver and spleen, and with many bone deposits. It ends fatally within weeks or months

Niemann-Pick's disease is another type of lipoidosis occurring in early infancy, characterised by the presence of sphingomyelin in the histiocytes. Skeletal lesions are rare

Lymphatic and myelocytic leukæmia—Bone changes often occur in lymphatic leukæmia and sometimes in myelocytic leukæmia. The lesions may be diffusely porotic and destructive, or there may be subperiosteal new bone formation. Diffuse porosis of vertebral bodies with spontaneous crush fracture resembles the changes of senile osteoporosis, but the bone changes of leukæmia usually occur in children rather than adults. The differential diagnosis is made easy by the blood cytology.

Lymphadenoma—Bone deposits in lymphadenoma are usually osteoclastic, resembling the metastatic deposits from carcinoma, but sometimes there is unusual density of vertebral bodies. Pathological crush fractures may be sustained.

PARASITIC DISEASE OF BONE CAUSING PATHOLOGICAL FRACTURE

Hydatid disease, though rare in Great Britain and the United States, still occurs with considerable frequency in the great sheep-raising countries, Australia, South America and South Africa, despite measures to improve the conditions of slaughter-houses and to prevent contact of domestic dogs with infested sheep.[1] The greatest frequency has been in Iceland where no less than one-seventh of the population were infected; in India and in China it is almost unknown. The fact that hydatid disease in man has almost disappeared in the British Isles, despite the occurrence of the disease among the twenty-five million sheep which are pastured, is attributable to strict regulations of abattoirs, inspection of meat, good water supplies, the standard of public hygiene, and care in feeding dogs. The tænia echinococcus inhabits the small intestine of the dog. A single dog may harbour thousands of mature worms and pass many thousands of ova daily in the fæces, thus contaminating food and water and infesting sheep or man—the intermediate hosts in which the larval stage of the worm develops with cyst formation. If dogs feed on the viscera of diseased sheep they become infested with scolices, of which there may be many thousands in a single fertile cyst, thus completing the life cycle of the tænia.

Clinical features—Man is usually infested directly from the dog which he pets, caresses or allows to lick his hand, the ova being resistant and surviving in dust for many months despite desiccation, soaking, heating and freezing. Primary cysts develop in bone in about 1 per cent of cases. They first appear near the epiphyses, usually in the femur or humerus, and less often in the vertebræ, tibia or pelvis. They grow so slowly over a latent period of many years and with such complete freedom from symptoms that the diagnosis is seldom established before adult life. Multiple irregularly shaped cysts closely resembling fibrocystic disease develop gradually, at first without bone expansion, but with steady infiltration along bony canals (Fig 700). Pressure absorption of bone, invasion of the cortex and periosteum which offer little or no resistance, and slowly progressive weakening lead finally and inexorably to spontaneous fracture. A fracture sustained without injury or with minimal injury is very often the first clinical manifestation of hydatid disease of bone. The fracture is painless because the nerves of the Haversian canals have been slowly and quietly destroyed. It is associated

[1] Dew, H R "Hydatid Disease Its Pathology, Diagnosis, Treatment" Sydney Australasian Medical Publishing Co, 1928

with little or no ecchymosis, swelling or œdema because the blood vessels have been destroyed and the bone is avascular. There is no crepitation, because the bone has been destroyed. Non-union is almost inevitable.

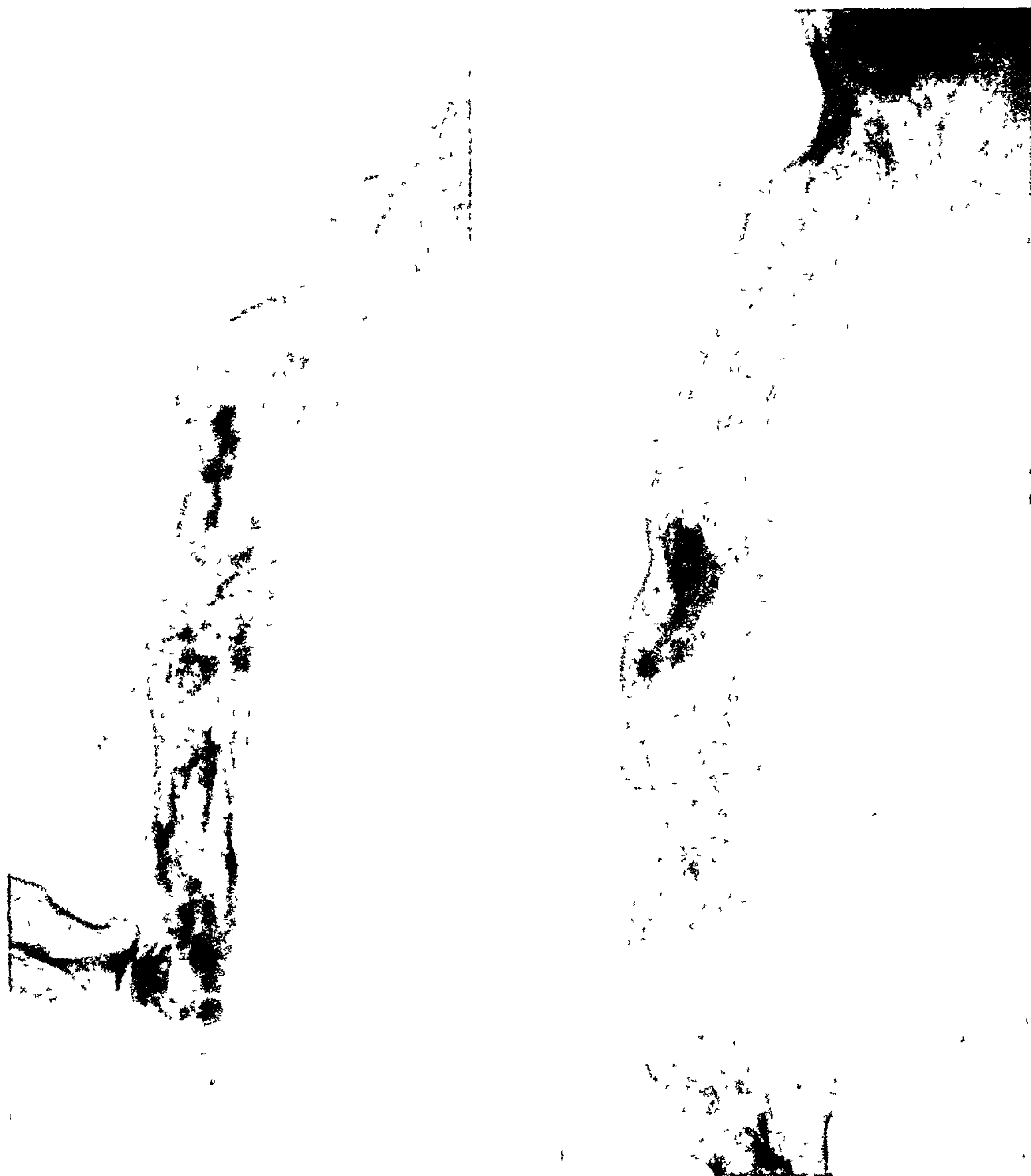


Fig. 700

Hydatid disease

Hydatid disease of the humerus in an African girl who sustained many fractures. In the last two years there has been improvement. Despite the bizarre appearance all fractures are united, but there is still gross infestation. (*Courtesy of Mr N. Teubes, Baragwanath Native Hospital, Johannesburg.*)

Within a period varying from two to ten months after fracture, as a result of the shedding of parasitic elements from bone into surrounding soft tissues, a large, cystic, painless swelling develops. There is little or no inflammatory reaction and the clinical picture may resemble a cold abscess.



Fig 701

Fig 702

Hydatid disease of bone

The first sign of disease was a pathological fracture of the femur. There is now complete destruction of the femur, hip joint and one-half of the pelvis There is no evidence of hydatid disease in the chest or elsewhere in the skeleton (*Courtesy of Dr W J Latham, Diagnostic X-ray Department, Groote Schuur Hospital, Capetown*)

Differential diagnosis—During the long period of latency, early cyst formation discovered during routine radiography is often mistaken for fibrocystic disease. When spontaneous fracture occurs, the bone destruction and total absence of periosteal reaction suggests secondary carcinoma. In the later stages of soft tissue dissemination, cold abscess is simulated. The diagnosis may be confirmed by the Casoni and complement fixation tests, and if necessary by diagnostic puncture. Microscopic examination of aspirated fluid shows fragments of laminated membrane, small daughter-cysts and sometimes scolices or booklets.

Treatment of pathological fractures in hydatid disease of bone—The prognosis is very grave because there is often non-union of the fracture, and extension rapidly occurs in soft tissues, neighbouring joints being involved; and there is considerable risk of pre-operative or post-operative infection which may be fatal (Figs. 701-702). No useful purpose is served by simple incision into the cysts, curettage or the application of formalin; these measures are almost invariably followed by recurrence. If the disease is still limited to bone and there is no invasion of soft tissues, total excision of the involved area followed by massive autogenous grafting is the treatment of choice. The excision must be sufficiently wide to include all cysts, however small. If soft tissues are already involved, amputation above the involved area is the only measure that offers hope, and it should be undertaken forthwith.

NEUROTROPHIC DYSTROPHIES OF BONE CAUSING FRACTURE

Tabes dorsalis and syringomelia—The degenerative process in the posterior columns of the spinal cord from syphilitic infection, and the peculiar cystic lesion of the cord in syringomelia, produce bone and joint changes which are indistinguishable. The neurological lesion destroys the sensory nerve tracts in the cord, and both joint sense and the sensation of pain in the affected areas are lost. Changes occuring in the joints were first described by Charcot [1] and are known by his name—Charcot's joints. The arthropathy is characterised by enlargement of the joint, effusion, laxity of ligaments, dislocation and subluxation, fracture, and deposition of new bone [2]. The characteristic feature of the lesion is that it is painless; gross joint changes and even fractures may occur without the patient complaining of pain (Fig. 706).

The bone and joint lesions were originally considered to arise from minor injuries sustained by structures unprotected by normal painful stimuli, but the more modern view, which is probably correct, is that they are the result of vasomotor trophic disturbances from involvement of the vegetative fibres in the spinal cord [3]. In this type of neuropathy bone does not behave like normal bone and bizarre complications may be encountered (Figs. 703-705). When there is neurotrophic disturbance of bone, union—either of a fracture or of an elective arthrodesis—is usually slow, and metallic internal fixation is not well tolerated.

[1] Charcot, J. M. "Sur quelques arthropathies." *Arch. de phys.*, 1868, 1, 1.
[2] Merritt, H. H., Adams, R. D., and Solomon, H. C. "Neurosyphilis." Oxford University Press, 1946.
[3] Biggart, J. H. "Pathology of the Nervous System." 2nd ed. Edinburgh, 1949.



Fig 703



Fig. 704



Fig 705

Fracture of femoral neck in tabes dorsalis

A patient with tabes dorsalis fractured the neck of his femur (Fig 703) It was nailed, and lateral and anteroposterior radiographs confirmed that the nail was accurately placed in the head of the bone (Fig 704) Two months later there was obvious redisplacement, but quite unlike the displacement often seen in this fracture from avascular necrosis of the femoral head, the point of the nail was still exactly central in the proximal fragment (this being confirmed in lateral radiographs not reproduced here). The nail had ploughed down through the neck and shaft of the bone until it reached the resistance of the calcar femorale on its inferior aspect (Fig 705) When there is neuropathic disturbance of bone, metallic internal fixation is not well tolerated (*London Hospital case*)



FIG. 706

Neuropathic dystrophy of bone—Charcot's disease

Patient with tabes dorsalis and Charcot's disease of the spine. The bone is most destroyed where it is most sclerosed—and this is the typical radiographic picture. Despite extensive bone destruction, causing wedging of vertebral bodies and scoliosis, there was no pain.

Bone lesions in diabetic neuropathy—That lesions at the ends of the extremities, usually the lower limbs, may occur as a complication of diabetes from vascular changes is well known. It is possibly less well known that diabetes may also cause a neuropathy which produces bone and joint changes similar to those seen in neurosyphilis and syringomelia This was first stated by Calvi [1] in 1864, and the subject has recently been fully reviewed by Rundles [2] This complication is distinguished from that of vascular degeneration by the fact that the lesions are painless Further, diabetic neuropathy does not occur as an acute hazard of diabetes, like acidosis,

[1] Calvi, M. de "Recherches sur les Accidents Diabétique" Paris P. Asselin, 1864.
[2] Rundles, R W "Diabetic Neuropathy" *Medicine*, 1945, 24, 111



FIG 707



FIG. 708



FIG 709

Neuropathic dystrophy of bone—Diabetes and peripheral neuritis

For twenty-one years this patient was treated for diabetes Although it was partly controlled by diet and insulin there was vascular disturbance in the feet and peripheral neuritis with impairment of sensation. A fracture of the neck of the second metatarsal bone was sustained five years ago, and a fracture of the third metatarsal shortly thereafter (Fig 707) These fractures united, but at an unknown date there had also been a fracture of the shaft of the fourth metatarsal, and six months ago there was a fracture of the neck of the fifth metatarsal (Fig 708) These fractures united, but there is now arthropathy of the first metatarso-phalangeal joint (Fig 709). (*London Hospital case*)

coma, or diabetic gangrene, but as the result of many months or years of uncontrolled or insufficiently controlled diabetes (Figs. 707-709). This, indeed, is a most important diagnostic point. The neuropathic bone or joint lesion is often preceded by paræsthesia, motor weakness, or other neuritic symptoms. The treatment depends essentially on expert diabetic management. If this can be achieved the prognosis is good, and fractures heal without difficulty. Joint disorders may, however, be permanent.

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